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University of Toronto

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Medical Journal

KATHRYN CHORNEY

- 1995 Gairdner Awards
- Pedicle Screws for Spinal Fusion
- Active vs. Passive Euthanasia
- Dehydroepiandrosterone
- Inferior Vena Cava Filters
- Football Cervical Spine Injuries

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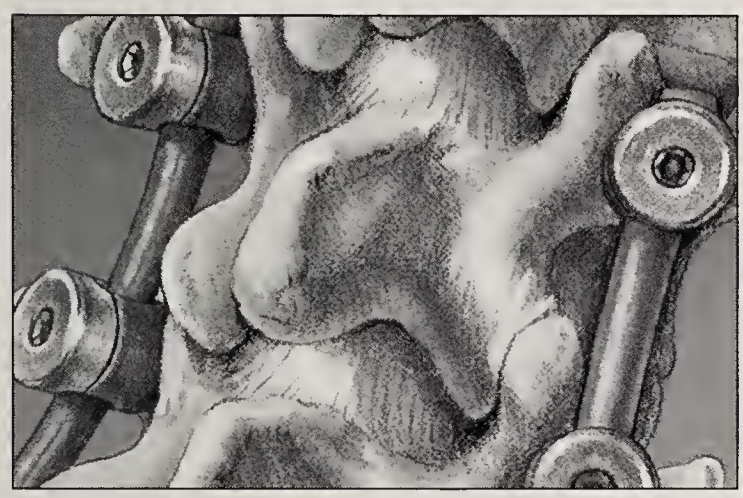

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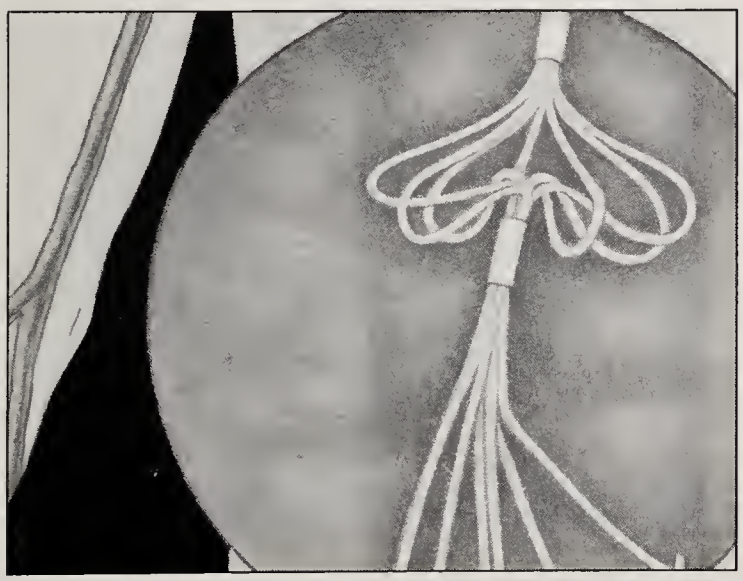

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Front cover illustration by Kathryn Chorney, Department of Biomedical Communications, University of Toronto.

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Editorial

The University of Toronto is world renowned for excellence in basic science and clinical research. In spite of diminishing financial support, the research community at U of T continues to flourish and generate work of the finest caliber. For seventy three years, the University of Toronto Medical Journal (UTMJ) has been a showcase for contributions made by scientists, clinicians and students at U of T to the existing body of medical knowledge. The Journal has evolved to become a publication of international stature with a circulation of thousands. It is in keeping with this tradition that we welcome you to the 73rd volume of this publication.

The UTMJ features submissions covering a wide range of topics. Research articles are supplemented by our *News, Commentary, Book Reviews,* and *Quick Diagnosis* sections. **For volume #73, we invite our readers to contribute to the Journal by submitting letters to the editors with comments and views regarding any of the UTMJ's contents or regarding any current topic in the field of medical science.**

Customarily, the *News* section of the UTMJ fall issue has featured the winners of the annual Gairdner Awards. Complementing this announcement, we have included an account of the much anticipated new Molecular Medicine and Human Genome Research Facility at the Mount Sinai Hospital. These two articles reflect the rapid progress that has been witnessed in the field of molecular genetics and the significant involvement of the U of T research community. Maintaining the focus on genetics, we have put forward in our *Commentary* section an essay that considers the uncertain role that the rapidly evolving field of molecular biology will play in the future of medicine.

The research articles in this issue cover an eclectic array of topics. Our featured submission describes the use of pedicle screws in spinal fusion. In the area of orthopaedics and sports medicine, a paper on cervical spine compaction associated with football injuries is presented. From the discipline of radiology, we include an informative article on the use of inferior vena caval filters. In the field of biochemistry, we present an intriguing paper on the possible effects on the aging process of the androgen prohormone dehydroepiandrosterone. In addition to submissions on surgery, medicine and basic science, we hope to reflect the spirit of intellectual discourse that exists in the U of T medical community regarding social and ethical issues as they relate to medicine. Pursuant to this idea, we are pleased to present a paper addressing the controversial and timely issue of active and passive euthanasia.

For those wishing to challenge their diagnostic acumen, we once again feature our popular *Quick Diagnosis* section. The current issue presents cases acquired from various surgical specialties. A prize of $50.00 will be awarded to the reader who submits the correct diagnosis for each of the cases presented in a particular issue of volume #73.

The UTMJ has earned its place among the most respected undergraduate medical publications in North America. It is through the continued support of our patrons that we are able to perpetuate the tradition of excellence associated with the Journal, and we gratefully acknowledge their contributions.

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NEWS

The 1995 Gairdner Foundation International Awards

Ilan Lenga (9T8)

On October 21, 1995, the Gairdner Foundation of Toronto, Ontario awarded three American scientists with their annual International Award for Achievement in Medical Science. Since the foundation's establishment in 1957 by the late James A. Gairdner, it has honoured 238 scientists. It is heralded as a "Nobel-predictor" as 45 of its honorees have subsequently gone on to become Nobel-laureates.

This year, the Gairdner foundation has recognized Arthur Kornberg and Bruce Alberts with a joint award for their work in the elucidation of the process of DNA replication. Dr. Kornberg is the third Gairdner winner to have been a previous Nobel winner (1959), joining the ranks of Frederick Sanger who received his Nobel in 1958 and his Gairdner in 1971, and H. Gobind Khorana awarded in 1969 and 1980 respectively. The other Gairdner honouree is Roger Tsien, who has won the award for his ground-breaking work on the design & synthesis of molecules which can measure minute amounts of intracellular chemical messengers.

Following two days of lectures and seminars at The University of Toronto, the three scientists received the $30,000 (Can) prize, with a framed inscription and sculpture, from Betram Brockhouse, himself a Nobelist in physics (1994), at an awards reception in Toronto.

Each year, the Gairdner Foundation International awards are given to a small number of scientists in recognition of their contributions to medicine.

Lifting the Fig Leaf

Co-Awardees Alberts & Kornberg have devoted the better part of their careers towards characterizing the events of DNA replication. Since Watson & Crick theorized a mechanism for DNA replication in 1953, continual efforts have been made to discover the process by which this replication occurs. Both scientists, in their lectures at UofT, joked about the replication-fork being traditionally obscured by a proverbial fig-leaf.

Dr. Bruce Alberts is being awarded specifically for his work on DNA replication using the T4 coliphage. Over the past 25 years his lab has intensively laboured to uncover the replication fork, and to characterize the many enzymatic players involved in the replication complex, which is able to copy both strands simultaneously as well as partially dissociate and re-associate when required. Alberts has had an illustrious career in the life sciences, and served as chair of the Commission of Life Sciences, National Research Council as well as president-elect of the American Society of Biochemistry and Molecular Biology. In 1980 he was awarded a Lifetime Research Professorship from the American Cancer Society. Alberts is principal author of the text *The Molecular Biology of the Cell*, the foremost textbook in its field used widely in North American universities.

Dr. Bruce Alberts

Alberts, 57, was born in Chicago, Illinois and attended Harvard until his post-doctorate appointment to the faculty of Princeton University in 1966. Ten years later he became professor and vice-chair of Biochemistry and Biophysics at the University of California, San Francisco (UCSF); he became chair of the department in 1985. Dr. Alberts now serves as the president of the National Academy of Sciences (USA).

Kornberg, 77, was born in Brooklyn N.Y., and obtained his M.D. from University of Rochester in 1941. He became a research assistant at the National Institutes of Health (NIH) in Bethesda in 1942, and rose to the rank of Medical Director by 1953. In 1959 he moved to Stanford University to organize and chair the biochemistry department for 10 years. He is currently active at Stanford in an emeritus position.

Dr. Arthur Kornberg

In 1959 Kornberg received his Nobel prize for identifying and purifying DNA Polymerase I from E. coli. Since that time his lab has discovered numerous enzymes and

co-factors many of which comprise the holoenzyme, a multi-subunit dimer which is the principal enzyme in rapid and high-fidelity DNA replication. Kornberg has also been honoured with membership in the National Academy of Sciences, the Royal Society, and the American Philosophical Society, as well as with the National Medal of Science (1979) among other medals and awards.

Kornberg considers himself an enzymologist in his scientific autobiography, *For the Love of Enzymes: The Odyssey of a Biochemist.* In his entertaining lecture at UofT, Kornberg professed his two deep faiths: in "enzymology being the core of biology" and in "the universality of biochemistry". These two faiths have allowed Kornberg to make important strides in basic research using E.coli and T4 Bacteriophage replication forks, with the expectation that these key enzymes have been conserved throughout evolution.

Molecular Spies

Dr. Roger Tsien

The work of Dr. Roger Tsien, for which he is receiving his Gairdner award, began as a "bootleg" project to his assigned graduate thesis[1] in which he designed and synthesized molecules which would change their fluorescence when bound Ca^{2+}. By injecting these molecules into cells it was possible to visualize changes in calcium ion gradients during the transmission of intracellular messages. In his lecture at UofT, Tsien played a video clip of the Ca^{2+} wave which travels through cardiac myocytes just prior to contraction. He referred to his method as "a marriage between old fashioned organic chemistry and new molecular biology". Since his original project Tsien has created molecules which fluoresce brightly enough to obtain recordings from single cells, and has synthesized other molecular spies for levels of cyclic adenosine monophosphate (cAMP), sodium ions, and hydrogen ions[1]. His work has immediate applications in many medical fields, including endocrinology, neurobiology, and immunology.

Tsien, 43, obtained his bachelor's degree in chemistry and physics from Harvard, and his doctorate in physiology from Cambridge. He served on the faculty at University of California - Berkeley, and in 1989 moved to UofC - San Diego where he now holds full professorship.

Along with his Gairdner award, Tsien has been awarded the international 1995 Artois-Baillet Latour Health Foundation prize, valued at $200,000 for scientists whose research has "immediate applications".

The Gairdner Foundation continues its committment to applaud the accomplishments of scientists such as these. In his lecture *The Ten Commandments of Replication Enzymology,* Arthur Kornberg said, "...and if I could add an eleventh, it would be 'Support Basic Research'". The Gairdner foundation appears to be doing just that.

UofT and the UTMJ continue to enjoy their special relationship with the Gairdner Foundation. A special thank you is owed to Verette Pennycook, the executive director of the foundation, for organizing the stimulating and insightful lecture series and for providing much of the information in this article.

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1. Sankaran N. Three Americans To Receive Gairdner Foundation International Awards. The Scientist 1995; 9(6): 1,9.

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Mount Sinai Hospital Building a New Molecular Medicine and Human Genome Research Facility to Study Human Disease

Sophia Hussain, B.A. (9T5)

Toronto, an international centre for genetic research, will be housing a new research facility which is expected to benefit the University of Toronto and attract leading international scientists.

The Samuel Lunenfeld Research Institute at Mount Sinai Hospital received funding from Britol-Myers Squibb Pharmaceutical Research Institute and the government to build a new Human Genome Research facility at the hospital. The building is to be named The Centre for Molecular Medicine and Human Genome Research. There, research will focus specifically on studying the genetic basis and molecular biology of human disease. It is considered one of the few centres of its kind in the world.

"The centre is important in the broader sense because it represents the directions we are heading which is the application of molecular medicine to research"

"The centre for Molecular Medicine and Human Genome Research is important in the broader sense because it represents the directions we are heading which is the application of molecular medicine to research," said Dr. Allan Bernstein, the Associate Director of the Samuel Lunenfeld Research Institute and Professor of Molecular and Medical Genetics at the University of Toronto.

The Samuel Lunenfeld Research Institute was established in 1985 and is considered to be one of Canada's top biomedical research facilities. The Institute is currently staffed by Dr. Bernstein, and colleagues doctors Tony Pawson, Andras Nagy and Janet Rossant, along with more than 60 international graduate students, post-doctoral fellows and research technicians. The Institute has four major areas of research: Cancer and Molecular Biology, Developmental and Fetal Health, Clinical Epidemiology, and Molecular Medicine. Research will also consist of subsequent diagnosis and treatment of human diseases such as breast and prostate cancer, heart disease, diabetes, bone disease, and neurodegenerative diseases once the new facility is available.

Senior scientist Tony Pawson, who received the Gairdner Foundation International Medical Research Award in 1994, has discovered, along with his research team, how cells communicate biochemically to control cellular growth and development. The team's work relates to Dr. Bernstein's research which involves determining the effect of communication breakdown on cancer cells.

Dr. Bernstein, who will be heading the Human Genome Research Centre, is also an internationally acclaimed scientist who earned his PhD in Medical Biophysics at the University of Toronto and completed a post-doctoral fellowship at the Imperial Cancer Research Fund Laboratories in London, England.

Bristol-Myers contributed $10.3 million which is to be distributed over five years to support the facility's medical research. In 1990, the company awarded $5.75 million to the Institute's scientists. In addition, the Canada/Ontario Infrastructure Works program, a joint program between metro, provincial and federal governments, approved $5 million dollars in funding last January.

Bristol-Myers support involves assisting the researchers in creating genetic mouse models designed to understand the causes of disease. "This is an important feature for molecular disease, which involves the use of gene targeting to make an animal model," said Dr. Bernstein. "The mice will provide the understanding of the disease and open up new opportunities for human testing."

Some of the benefits of the new centre include filling 150 new full-time and permanent research positions, acting as a catalyst for genome research internationally, and providing technology transfer for new diagnostic and therapeutic products, which will enhance the population's health care delivery. A unique multilaser facility for use by the pharmaceutical industry and the research community will also be available.

According to Dr. Berstein, the centre is also expected to benefit the University of Toronto in several ways. First, the centre will provide a vehicle for introducing research results in the clinic, for example, cloning disease genes which could enable testing on human DNA. Second, part of the new centre will deal with ethical issues and recruit investigators to address issues concerning medical technology. Third, the centre will benefit the University of Toronto students by providing research facilities for new and original initiatives using leading edge technology.

The commitment of Bristol-Myers Squibb has been vital to the scientists at Samuel Lunenfeld Research Institute to maintain our leadership frontiers of medical research," said Dr. Bernstein. "With the company's renewed support, we hope to continue to attract the very best young people from Canada and around the world to this research and work toward answers to important questions in biology and medicine.

Construction is scheduled to begin in October 1995 and the facility is expected to be completed in July 1996.

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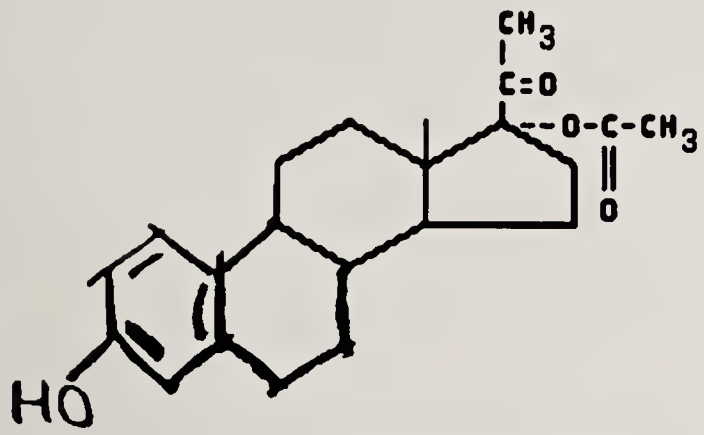

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COMMENTARY

Is Molecular Biology the Future of Medicine?

Emmanuel Farber, M.D., Ph.D., F.R.S.C.

Department of Pathology, Anatomy and Cell Biology, Jefferson Medical College, Philadelphia

The public perception of the beneficial role of science to our health and to the control of disease has been increasingly positive over the past few decades. In medicine and biology, the area of science receiving the most attention is molecular biology, that branch of biochemistry that is concerned mainly with molecular genetics and the functioning of the genome. An increasing number of physicians in all areas of medicine including many in pathology and dermatopathology believe that molecular biology will revolutionize our concepts of disease and the way we practice medicine. Since, as yet, there has been almost no impact of molecular biology on the diagnosis and treatment of most diseases, the conceptual belief in the impact of molecular biology in medicine relates, of necessity, to the future. Thus, this view is expressed largely in terms of hope, not reality.

This orientation is particularly common in those areas of medicine that pose our greatest challenges to their prevention and cure - cancer, arteriosclerosis and its many consequences, Alzheimer's disease, multiple sclerosis, amyotrophic lateral sclerosis, psoriases, etc. How realistic is this projection? Should the future physician become a molecular biologist, so as to be ready when the revolution comes?

As some of my colleagues and friends know, I started my career in medicine and medical science with the strong conviction that biochemistry was "the way to go" and that molecular biochemical approaches offered the possibility for answers to many of the problems in disease. My experiences over the past few decades have forced me to progressively change my mind. I now know that the approaches used in biochemistry-molecular biology which we employed early in my career, and which are now being used to an increasing degree, are inappropriate if we wish to understand mechanisms of disease and if we hope to develop rational and effective disease preventions and cures. The old-new approaches will no doubt generate a plethora of publications and will hasten advancement in the academic ladder, but won't help us much in our quest for understanding the dynamics of disease development and favor the developing of innovative new approaches to disease control.

Why are the prospects for molecular biology in pathology and medicine not more positive? This limitation is based on my increasing awareness of the current misconceptions in biology and medicine, and of a more realistic view of how the host responds during disease development.

It is regrettable that many physicians still view the human being as essentially a mechanical construct and that disease is largely the result of wear and tear of the parts of the machine. The cells and tissues are often considered to be static structures and disease viewed, naturally, as being due to the destruction and/or abnormal function of one of the parts of the machine. In other words, disease is reviewed as being the result of the passive "pushing and shoving" of cells and tissues by some agent or agents of external or internal origin.

Of course, we now know that every living organism and every living part of an organism is extremely dynamic in that genes, immediate gene products, cell membranes, mitochondria, endoplasmic reticulum, etc., etc. are undergoing change continuously, yet the organism and its parts are able to maintain continuity and stability.

"...physicians in all areas of medicine...believe that molecular biology will revolutionize our concepts of disease and the way we practice medicine...this view is expressed largely in terms of hope, not reality."

Since each specific piece of information, i.e., each protein and each precursor or product of protein function, is coded for in the genome, it is easily understandable that a simple-minded highly focused approach, with the best of intentions, would concentrate on the gene as the site for all biological activity including disease. The truly remarkable radical changes in technology in the past two to three decades have offered seemingly endless possibilities. Identification and isolation of genes, the immediate gene products, messenger (m)RNA and the ultimate gene products, the proteins, are being accomplished daily. Receptors on the cell membrane and inside cells are being identified and purified with increasingly efficiency. The continual recycling of membrane and other cell components between the outside and the inside and vice versa is being studied with increasingly precision.

To accomplish these feats, increasing numbers of young scientists, mainly non-medical, are being enticed into cell and molecular biology. Included are many budding scientists who are engaged in studies of disease. They are generating pieces of information at an every expanding rate. The apparent success of this enterprise is rapidly causing a new concept of disease: that the acquisition of the bits of information will in and of itself offer and explain these disease mechanisms. We do not have to know or understand "old fashioned biology" - just do enough studies of genes, mRNA's, proteins, receptors, etc. etc., and everything will fall in place. This

expanding enterprise in disease is being accepted, superficially, by many pathologists, as witnessed by the papers and posters at national pathology meetings and by the type of publications in "reasonably good" current pathology and other medical journals. This approach, if pursued vigorously, could well bypass the scholarly practicing pathologists, since they are not truly in the massive data-collecting operation and therefore would be essentially useless.

Stating the problem somewhat simplistically. all we have to do to solve any complex jigsaw puzzle is to collect as many of the pieces we can and then simply let them fall at random. Somehow, the pieces will find their appropriate neighbors automatically and solve the puzzle. This would apply to puzzles with thousands of pieces of information as occurs in living cells and tissues, as well as in puzzles with only a few pieces.

The fallacy of this approach is evident when we begin to examine it in a little more detail. Two simple-minded examples among many can be mentioned. In the first, it has been repeatedly pointed out that the DNA from chimpanzees, gorillas, and humans show 98% or more homology, i.e., our DNA is ostensibly very similar to that of chimpanzees and gorillas. Yet somehow, I have absolutely no difficulty in easily identifying chimpanzees and gorillas and especially humans when I visit the zoo. If all our music, education, art, mechancal dexterity and accomplishments, etc. etc. depend only on a 1 to 2% difference, I say that DNA homology is not a very potent tool in biology, even though it is the basis for many of the modern DNA technologies. In the second example, I find no difficulty in readily distinguishing brain, bowel and bone marrow, even though current DNA technology tells me that are all the same. Again, the variations in DNA structure, i.e., the polymorphism, is most impressive. With 4 to 5 billion humans, each with differencies in the DNA, how do we reconcile each being human with our curent simple view of the genome?

Clearly, what I am saying is that all structure and function in an intergrated cell, tissue and human, depends upon patterns of gene expressions, as reflected in patterns of protein presence and function, not on the presence or altered presence of genes themselves. Obviously, with no gene, there is no expression. However, cells are enormously talented in the way they orchestrate their gene expression during their many different biological behaviours.

Medicine and pathology are based on differences in patterns and in pattern recognition, i.e., on patterns of gene expressions and the responses of cells, tissues and the whole person to such patterns. Obviously, we must know what the cells and tissues have the capacities to do, but we must also know how the totality is put together.

In this context, we can liken current cell biology and pathology to the differences between an alphabet and a language. With a relatively simple alphabet like the Latin alphabet, with only 26 letters, we can theoretically produce thousands of languages. Even with the few Latin-based languages we currently use, knowing the alphabet does not enable us to know each of them automatically.

The pathologist, as the most interested participant in pattern recognition at the tissue and cellular levels, is absolutely essential if we are to use intelligently the innumerable bits of information generated by molecular biology. To do this however, we must become very familiar with how the bits of information were generated and with how cells and tissues shuffle these bits in infinite ways to express a wide variety of biological behaviors.

In addition, there is another major dimension that we must emphasize in our roles in both practice and research, namely response patterns. Much of what we see in disease is not the direct result of some etiological agent or perturbation, but rather the response patterns that the living cell, tissue and person makes during the disease process. This is seen in every disease, both acute and chronic. In many acute diseases, some of these response patterns have been emphasized by Wiley Forbus in his book "Reaction to Injury"[1].

The key roles of the response patterns are ever more evident in chronic diseases that may require many years or decades to develop. The importance of the adaptive response patterns has been emphasized by many thoughtful and scholarly pathologists for many years[e.g.,2-4]. I consider that the long process of cancer development with chemicals, DNA viruses and radiations is largely a process of adaptation that could initially be beneficial to the organism, and the disease component, the cancer, may be an expression of the ultimate failure of the adaptive process[5,6].

The pattern recognition, response patterns and adaptive patterns are not likely to be meaningful in current molecular biological terms, even though they probably are absolutely critical to our understanding of disease. Unless molecular biology, in a radical new orientation, can offer us some new insight into how we should study these responses of the host in disease, it is not evident why we should generate the obeisance that is demanded of us by the single-minded molecular biologist.

Dr. Emmanuel Farber was the Chairman of the Department of Pathology at the University of Pittsburgh from 1960 to 1970. He returned to his alma mater, the University of Toronto, as Chairman of the Department of Pathology from 1975 to 1986. From 1975 to present he has held the position of Professor of Patology and Biochemistry. He is currently Professor of Pathology, Anatomy and Cell Biology at Thomas Jefferson Medical College. His main research interests are the biochemistry-molecular biology of basic disease processes including cell death and cancer development with chemicals. He has received international recognition for his contributions to the scientific analysis of disease.

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The Use of Pedicle Screws in Spinal Fusion: FDA Approval and Current Controversy

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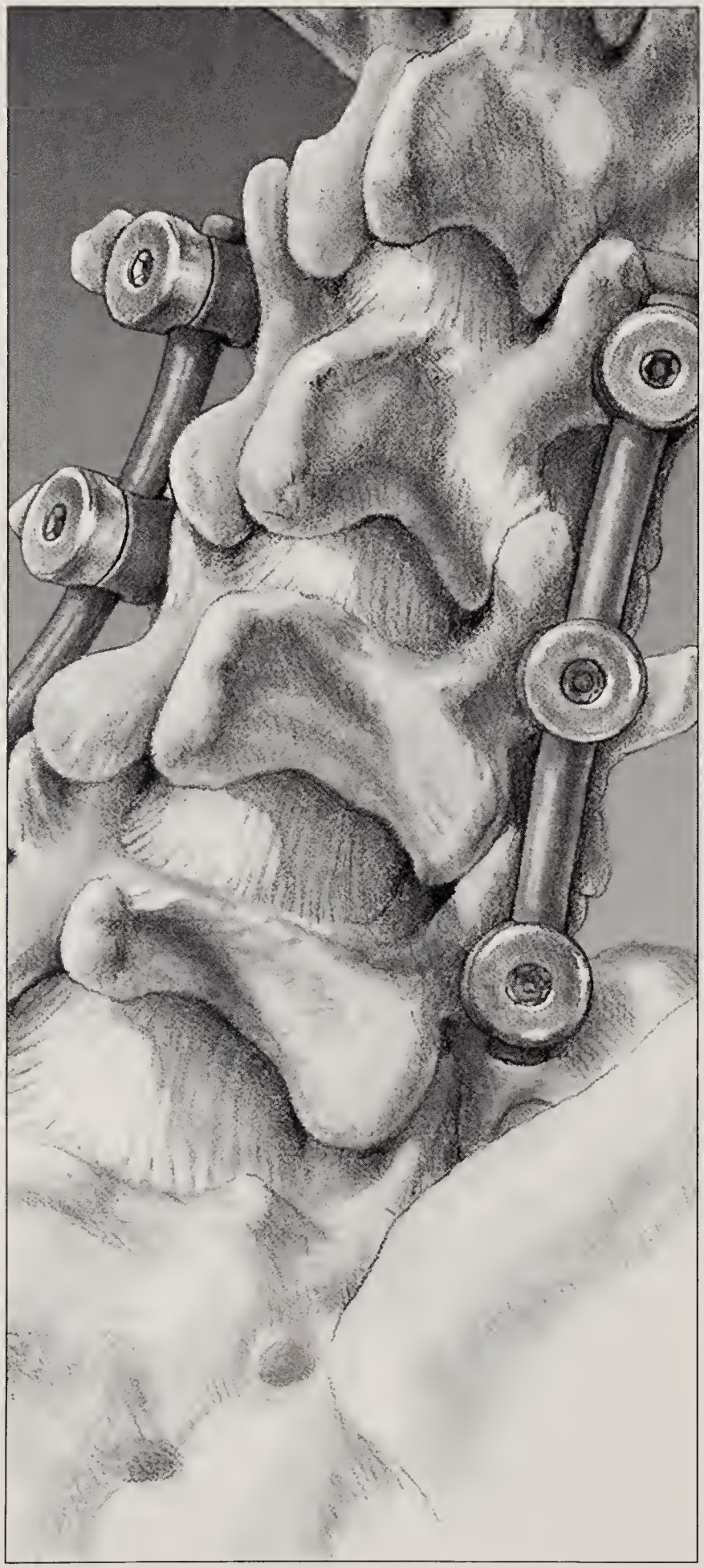

KATHRYN CHORNEY

Abstract

There are many painful conditions for which spinal fusion may be considered. Most cases of back pain, however, can be managed conservatively, and spinal fusion is considered only after failure of alternative treatment. More recently in spinal fusion surgery, a fixation technique that uses screws driven through the pedicle into the vertebral body has been used in conjunction with bone graft to achieve spinal segment immobility. While pedicle screw systems have many advantages over other fusion techniques, pedicle screws are not yet FDA approved and have recently been the subject of controversy in the media. Debate over the use of pedicle screw systems often confuses two separate issues: (i) the efficacy of pedicle screws in comparison to FDA approved instrumentation, and (ii) the controversial issue over indications for spinal fusion surgery. This paper reviews the indications, complications and efficacy of spinal fusion with pedicle screws, and elucidates the current issues and concerns raised by the media. A review of current literature highlights the advantages of pedicle screw systems, and supports that they are safe and effective instrumentation for spinal fusion.

Introduction

Clinical use of spinal arthrodesis was first reported in 1911[1,2]. Many surgical procedures have since evolved, and the indications for spinal fusion have been broadened. Fusion of mobile spinal segments is most commonly performed for treatment of trauma, spondylolisthesis, tumour, deformity, and degenerative arthritis[3,4]. In most cases, however, spinal fusion is a purely elective procedure, undertaken only when a patient is unable to cope with his or her pain and after failure of conservative treatment. While pedicle screw systems are frequently used to immobilize the spine in fusion surgery, they are currently not approved by the Food and Drug Administration (FDA) in the USA for their intended use in spinal fixation. The media have recently been disseminating the current FDA status of pedicle screws, and have generated controversy over their safety and efficacy[5-8].

The aim of spinal fusion for treatment of conditions such as degenerative disc disease and spondylolisthesis is to stop

motion of the intervertebral discs and the facet joints, thus reducing the amount of pain generated from these structures. Figure 1 illustrates the lumbosacral spine with pedicle screws in place and bars rigidly connecting the heads of each screw. Alternatively, arthroplasty may be suggested for treating painful mobile joints; however, clinically useful arthroplasty for the spine is still in the developmental stage[9].

There are several surgical techniques currently in use for promoting spinal arthrodesis[10]. In common with each technique, and the most important determinant of fusion, is the use of bone graft to achieve biological fusion of affected vertebrae[11]. While fusion may be accomplished with bone graft alone, the use of instrumentation increases rigidity at the fusion site, increases the fusion rate, and maintains intervertebral alignment[11,12,13].

When considering the success of spinal fusion surgery, it is important to distinguish between rate of successful bone growth and biologic fusion, and the comparatively lower rate of patients who experience some relief of pain and improved functional ability. Many patients with a solid arthrodesis have persistent symptoms and consider fusion surgery to be a failure[14]. Turner *et al* (1992)[14] reviewed 47 studies on spinal fusion and determined that success, being defined as some improvement in pain and function, occurs for an average of 68% of spinal fusion patients[14]. Note that the rate of clinically satisfactory outcome can vary significantly with study methodology, diagnosis, and spinal fusion technique[14,15]. In addition, compensation status and psychological disturbance are significant prognostic factors in fusion surgery outcome[16]. Length of time off work, age at time of injury, sex, social group, and a history of pain onset after slipping or jerking injuries are also important prognostic factors in recovery from back pain[17]. It is important to note that clinical success does not necessarily imply absence of pain and complete return to function; clinical success may also include conditions of mild persistent or occasional moderate pain, and restrictions on performing some normal activities[14].

Indications for Spinal Fusion

It is estimated that 50% to 70% of adults report having experienced low back pain at some time in their lives, and in the USA, less than 2% of adults have any type of back surgery[18]. Spinal fusion is thus not a common procedure for managing back pain, and the indications vary depending on the type and severity of a condition. In cases of instability from trauma or degenerative scoliosis, there is generally agreement on the appropriateness of spinal fusion[3]. It is also a generally accepted treatment for more severe cases of spondylolisthesis to correct vertebral instability and to relieve pain[3]. For cases of nerve root compression, surgical treatment is commonly a laminotomy and discectomy, without fusion, to relieve pressure on the nerve root. When the compression of neural structures is more severe, as in spinal stenosis, large portions of the lamina and facet joints must often be removed. Since the facet joints are important load-bearing structures[19], removal of significant portions of the facet joints can result in iatrogenic instability and spinal fusion may be required[20].

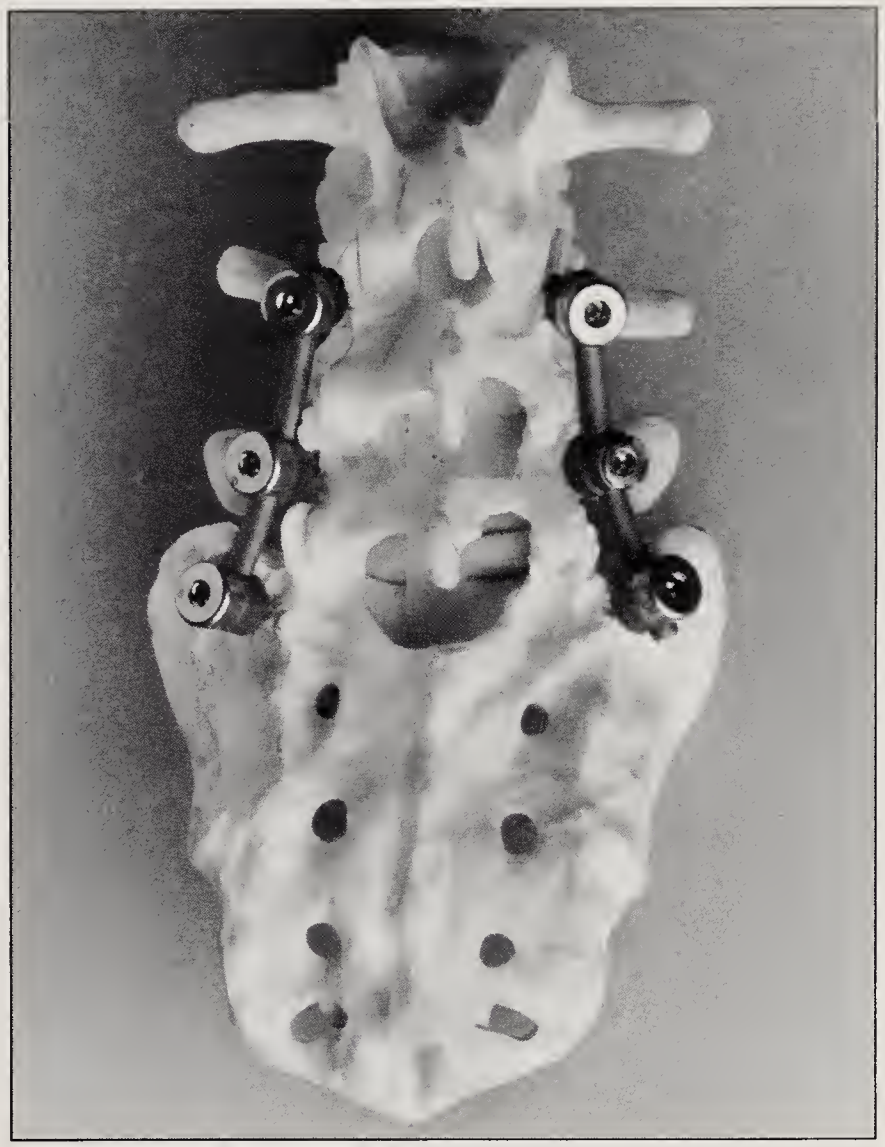

Figure 1: *Posterior view of the lumbosacral spine with pedicle screw instrumentation from L4 to S1.*

Much of the debate on whether or not spinal fusion is appropriate treatment commonly centres on its use for treating back pain associated with degenerative lumbar disc disease[21,22]. Back pain may arise from degenerative changes occurring in the intervertebral disc[23,24], from facet joints[19], or from compression of spinal nerves or roots[24]. The intervertebral discs in every person develop age-related degenerative changes which may lead to problems such as disc herniation or facet joint degeneration. The rate and extent of disc changes, however, vary from person to person and are also variable among the discs within an individual[23]. For the treatment of degenerative disc disease, there are currently no general guidelines for the suitability of spinal fusion. Furthermore, while it has been shown that in the long term a majority of patients with lumbar fusions do not experience severe pain, although more than the general population[25], there have been no studies evaluating lumber fusion in comparison to conservative treatment alone. In general, spinal fusion to immobilize painful segments should only be considered for patients with chronic disabling pain, and for whom conservative therapy has been a failure. Patients must appreciate that surgery for degenerative disc disease is an elective procedure, and must carefully evaluate their pain and functional disability given the success rates and measures of success discussed earlier. The

Figure 2: *Lateral radiograph of an L4 to S1 fusion with pedicle screws for treatment of degenerative disc disease (3 days post-operative).*

decision to proceed with surgery is further complicated by a patient's psychological and social attributes which, in cases of chronic pain, may become more of a determinant of functional status than nociception[26].

Use of Pedicle Screw Systems

Surgical Procedure

Fusion of the lumbar spine may be accomplished by a number of approaches and internal fixation devices. With a transpedicular screw system, a common posterior approach is used, beginning with a longitudinal midline incision through the skin, subcutaneous layer, and thoracolumbar fascia. The supraspinous ligaments are cut, and the paravertebral muscles are stripped and retracted from the spinous processes laterally to expose the remainder of the spine out to the transverse processes. The soft tissues of the facet joints to be fused are removed, and the spinous processes may also be excised to contribute to the bone graft. A decompression laminectomy is performed if required, and the surrounding transverse processes and pars interarticularis are decorticated in preparation for bone grafting. The posterior end of the pedicle is then located, and a probe is inserted through the pedicle and down to the anterior cortex of the vertebral body. The depth of the pedicle and vertebral body is then measured and a self-tapping transpedicular screw is then inserted. Once all pedicle screws are in place, rods or plates are used to rigidly connect the heads of the pedicle screws. An intraoperative radiograph is then taken to ensure alignment of the pedicle screws, and bone graft taken from the iliac crest and the spinous processes is subsequently packed into the facet joints and around the decorticated areas of the vertebrae. Figure 2 shows a lateral radiograph of the lumbosacral spine, with the same pedicle screw instrumentation illustrated in Figure 1. The AP radiograph in Figure 3 illustrates the fused spine one year post-operative; note the outline of the consolidated bone graft along the transverse processes.

Efficacy

Studies evaluating the effectiveness of spinal fusion for improving pain and function demonstrate a wide range of results. Recall that assessment of clinical outcome is dependent on diagnosis, study methodology, definition of success[14,15], compensation status, and other factors[16,17]. It is thus not surprising that studies evaluating clinical success after spinal fusion show a wide range of results and do not consistently identify any technique as yielding a better functional outcome[12,14,27,28].

As with studies evaluating the clinical success of fusion surgery, the published rates of fusion are also variable[14,15,28,29]. Yahiro (1994)[27] performed a ten year literature review of fusions with pedicle fixation and evaluated an overall fusion rate of 94.8%. However, the rate of pseudarthrosis, or failed fusion attempt, is affected by the number of segments fused and the method of diagnosis. Significant discrepancies have been found between radiologic findings of pseudarthrosis and routine second-look procedures[29]. It is therefore not possible to make accurate comparisons among the published rates of fusion. There are, however, numerous advantages of pedicle screw systems over the conventional FDA approved Class II instrumentation and over fusion without instrumentation.

A unique advantage of a pedicle screw system is that it provides rigid fixation for any loading condition applied to the spine[30]. With alternative internal fixation devices, there remains the possibility for relative motion between segments during the critical time that the bone graft is incorporating. Pedicle screw fixation also allows for short-segment fixation with preservation of lumbar lordosis[13,31]. In addition, all metal is kept out of the spinal canal with pedicle screws, and they may be used relatively safely when laminectomy has been performed, which is not the case for most other systems[28]. Finally, sacral fixation is readily performed with pedicle screws, but is difficult to achieve with conventional hooks[31].

Yuan *et al* (1994)[13] conducted a recent cohort study of pedicle screw fixation for thoracic, lumbar, and sacral fusions, collecting data from 3498 cases of degenerative spondylolisthesis and spinal fracture. It was determined that for degenerative spondylolisthesis, the pedicle screw systems had an overall better performance than a control

group of fusions without instrumentation (a control group of fusion with conventional instrumentation had too few cases for statistically significant comparisons). For cases of fracture, pedicle screws were found to have comparable safety and efficacy to other internal fixation devices, and for the same number of affected vertebrae, fewer motion segments were fused. It was concluded that "the use of pedicle screws is at least as safe and effective as conventional forms of spinal fusion surgical treatments for the two diagnostic indications studied" [13].

Complications

Pseudarthrosis is typically the most common complication of fusion surgery[14,29], and as discussed earlier, has a high degree of uncertainty in the published rates of occurrence. The ten year literature review of pedicle fixation by Yahiro (1994)[27] found that complications included broken pedicle screws (7.1%), broken rods (0.2%), malpositioned screws (2.5%), neural injuries or neurologic deficits (1.7%), and dural tears (1.1%).

Note that the pedicle screw literature review[27] incorporates studies published throughout the past ten years; pedicle screw systems and surgical techniques have since continued to advance. While the study by Yuan *et al* (1994)[13] reported screw breakage of 2.6% for spondylolisthesis and 6.7 % for vertebral fracture, the incidence of neural and vascular injury were each less than 0.5%. In an FDA approved clinical trial of pedicle screws, Steffee and Brantigan (1993)[15] published that screw breakage occurred in 2.5% of the 1314 implanted screws. A recent study by Lonstein *et al* (1994)[32] reviewed the complications of 1999 screws placed in 405 patients and found that only 0.9% of screws broke. In comparison to the rates of pedicle screw breakage, use of conventional Harrington distraction rods have a hook dislocation rate of 5% to 10% [33]. Furthermore, dural tears are more common with conventional Class II devices as these instruments intrude into the canal against the dura[28].

Other commonly reported complications of fusion surgery in general include persistent pain at the bone graft donor site, infection, deep vein thrombosis and pulmonary embolism[14].

Current Controversy over Pedicle Screw Fixation

The use of the transpedicular screw was first reported in 1969, and many advances in transpedicular screw-based devices have since been developed[33]. The FDA, however, still classifies these devices as Class III[28]. That is, pedicle screws are approved for use in long bones, but are not approved for their intended purpose of transpedicular spinal fixation. Recently, media attention to the current FDA status of pedicle screws has generated controversy over their safety and efficacy[5-8]. The Public Citizen Health Research Group, consumer advocates focusing on health, charge that the unapproved use of pedicle screws in spine surgery has resulted in complications such as blood vessel injuries, nerve damage, and bowel and bladder problems in up to 33% of cases[5,6]. An episode of ABC News 20/20[7] stated in 1993 that "...the screws, as first developed in the early eighties, did often break, nearly one in three, according to one early study. They broke so often they had to be repeatedly redesigned...". The North American Spine Society, however, maintains that current evidence supports that pedicle screws are safe and effective in certain cases[5].

A review of References 5-8 demonstrates that the media have been confusing two separate issues: (i) the current FDA status of pedicle screws, and (ii) the indications and complications of spine fusion in general. There is no doubt that the patients interviewed by the media[6,7] have genuinely experienced a great deal of pain associated with fusion surgery or its complications. The media, however, are presenting cases where spinal fusion has been unsuccessful, complications were encountered, or perhaps should not have been performed, and have confused it with the safety of pedicle screw systems. As discussed earlier, many surgeons will debate the efficacy of spinal fusion for treatment for conditions such as degenerative disc disease. The use of current pedicle screw systems, however, appears to be at least as safe and effective as FDA approved Class II systems.

Consider first the issue of pedicle screw fracture, which has been used to highlight dangers of the unapproved back screws[6,7]. Recall that the overall incidence of screw fracture in a ten year literature review was found to be 7.1%[27], and more recent studies have demonstrated screw breakage rates of less than 2.5%[15,32]. With spinal fusion surgery,

Figure 3: *AP radiograph of the same fusion in Fig. 1, but shown 1 year postoperative.*

instrumentation should be viewed as a temporary measure while the bone graft is incorporating[31]; the bone graft eventually becomes the long-term load bearing structure. Cases of pseudarthrosis, for instance, allow continued high stress cyclic loading of the screws and must be treated early to reduce the risk of fatigue failure or implant loosening[34]. Furthermore, conventional Harrington distraction rods have a hook dislocation rate of 5% to 10%[33]; the instrumentation failure rate with pedicle screw systems is no higher than the currently FDA approved Class II devices[28]. Highlighting cases of pedicle screw fracture merely highlights some of the common complications of spinal fusions with instrumentation.

Consider next the issue of screw misplacement. The Public Citizen Health Research Group has been emphasizing a frightening rate of damage to neural and vascular structures of up to 33%[5,6]. Studies performed using axial computed tomography (CT) to determine screw misplacement publish misplacement rates ranging from 0% to 40%[35]. A recent study by Botsford *et al* (1995)[35] has addressed the accuracy of axial CT in the assessment of screw misplacement using three dimensional CT reconstructions. It was determined that axial CT overestimates the number of screws that breach the pedicle cortex. It was also noted that apparent rate of screw misplacement varies with the implant material; use of stainless steel implants causes a substantial amount of artefact on axial CT in comparison to titanium alloys[35,36]. In addition to the CT studies on screw misplacement, the ten year literature review of pedicle fixation[27,28] found that malpositioned screws occurred in only 2.5% of cases, and the overall rate of neural injury was 1.7%. Recent studies have reported the incidence of neural injuries to be less than 0.5%[13,32]. In comparison, a literature review of spinal fusions where only 36% of the cases reviewed had instrumented fusions of an unspecified types, found the average incidence of neural injury was 2.8% with a range of 0% to 16.6%[14]. Finally, the recent cohort study of pedicle fixation confirmed that pedicle screws have no higher complication rates and no more significant complications than conventional Class II instrumentation[13,28].

Conclusions

The current controversy over the use of pedicle screw systems appears to be more regulatory than scientific in nature[4,28]. While the efficacy of pedicle screws for treatment of degenerative disc disease has not yet been studied, pedicle screw instrumentation has been shown to be at least as safe and effective as FDA approved Class II systems for the treatment of fracture and spondylolisthesis[13]. Pedicle screws also offer numerous advantages over the conventional Class II instrumentation[13,21,30,31]. Currently, spinal fusion with pedicle screw fixation is commonly performed for treatment of degenerative disc disease[3]. The issue of pedicle screw safety and efficacy, however, is often confused with the contentious issue over the use of spinal fusion for treatment of degenerative disc disease[21,22].

The media presentations of pedicle screw systems highlight their status of 'not FDA approved' for use in the spine, and often present a small number of cases where complications were encountered. In addition, the rates of complications with pedicle screws stated in the media productions far exceed those of comprehensive literature reviews and current studies. The public is further misled by the media reports which evade the issue of the indications for spinal fusion, and also omit the typical success and complication rates for any type of spinal fusion procedure.

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Active vs. Passive Euthanasia: The Importance of Context

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Abstract

A recent case involving a surgeon from Timmins, Ontario, turned on the distinction between passive and active euthanasia – that is, between letting patients die by withholding treatment and taking active steps to bring about their death. The decisions of both the court and the disciplinary committee of the Ontario College of Physicians and Surgeons were based on the assumption that while passive euthanasia may be permissible in certain circumstances, active euthanasia is never permissible. This doctrine was explicitly endorsed by the Canadian Medical Association in a policy summary on physician-assisted death published earlier this year.

In this paper, I outline the arguments of two philosophers, James Rachels and Philippa Foot, on the question of whether there is, in fact, an important moral difference between killing and letting die. I then argue that the moral implications of active and passive euthanasia vary from one context to another, and so our policies on euthanasia should vary accordingly.

Un cas récent concernant un chirurgien de Timmins, en Ontario, a mis en évidence la distinction entre l'euthanasie passive et active, c'est-à-dire entre laisser mourir un patient en ne lui administrant pas de traitement et prendre des mesures pour hâter le décès. La décision de la justice comme celle du comité disciplinaire de l'Ordre des médecins et chirurgiens de l'Ontario est basée sur le principe que si l'euthanasie passive peut être permise dans certains cas, l'euthanasie active ne l'est jamais. Cette doctrine a été clairement sanctionnée par l'Association médicale canadienne dans une publication parue cette année sur les règles qui gouvernent l'aide médicale à la mort.

Dans ce rapport, j'ai passé en revue les arguments de deux philosophes, James Rachels et Philippa Foot, portant sur la question de savoir s'il y a une différence morale sérieuse entre tuer et laisser mourir. J'argumente alors que les implications morales de l'euthanasie active et passive varient d'un contexte à l'autre et que nos politiques sur l'euthanasie doivent par conséquent varier également.

On October 15, 1991, Mary Graham, dying from lung cancer and on a respirator at the former St. Mary's Hospital

in Timmins, Ontario, told her three sons that she wanted the breathing tube removed from her mouth so that her suffering could come to an end. They supported her decision and conveyed it to her surgeon, Dr. Claudio Alberto de la Rocha. In accordance with standard practice, Dr. de la Rocha removed the tube and administered a total of 40 milligrams of morphine in three doses to ensure that Ms. Graham did not experience a suffocating feeling. He broke with standard practice, however, when he then gave her an injection of potassium chloride, causing her heart to stop.

In 1993, Dr. de la Rocha was tried in criminal court for administering the potassium chloride. He was convicted under Section 245 of the Criminal Code, which makes it an indictable offence to administer a noxious substance,[1] but his sentence was suspended. Earlier this year, Dr. de la Rocha had a hearing before the disciplinary committee of the Ontario College of Physicians and Surgeons. He was charged on two grounds: first, that his suitability to practise medicine had been compromised by his criminal conviction, and second, professional misconduct for administering the potassium chloride. Dr. de la Rocha had little choice but to plead guilty to the first charge, and when he did, the College decided not to proceed with the second charge. His penalty was a 90-day suspension of his licence that would be lifted if he wrote a guideline, intended for use in community hospitals and of a quality suitable for publication in a peer review journal, on how to withdraw life support from terminally ill patients. Dr. de la Rocha agreed to write the guideline and is presently working on it.

It should be noted that both the court and the College skirted the obvious issue in this case – euthanasia. There is no legislation in Canada that gives a person authority to take the life of another, and no exception is made for euthanasia[2] So instead of being charged with murder, which obviously would have been inappropriate, Dr. de la Rocha was charged with the far lesser offence of administering a noxious substance. The College, for its part, has no official policy on euthanasia, but had it proceeded with the charge of professional misconduct against Dr. de la Rocha, the ruling probably would have established one. Instead the College took the easy way out, penalizing him on the basis of his criminal record. Strangely enough, the only person involved in the case who has to tackle the difficult and emotional issue of euthanasia is Dr. de la Rocha himself, for he must now write a guideline on withdrawing life support that addresses not only the pharmacological management of symptoms, but also the following topics: informed consent, patient autonomy, the roles of the family and the family physician, and ethical implications. It might be said that the College's handling of the case is like punishing bank robbers by having them write the laws on theft.

Apart from the absurdity in the way the case was handled, there is also a certain absurdity in the case itself. It was captured nicely by Niels Ortved, Dr. de la Rocha's lawyer, in a statement he made in his final argument before the College's disciplinary committee: "The error was in the choice of drug. If morphine [alone] had been used, none of us would be here."[3] This statement also reveals that the central issue in the case is not so much whether euthanasia is right or wrong, but whether there is an important moral difference between passive and active euthanasia – that is, between letting patients die by withholding treatment and taking active steps to bring about their death. Obviously, both the court and the College maintain that the difference *is* morally important: one may remove a breathing tube from a patient's mouth; one may administer morphine to relieve suffering, even though the drug might also depress respiration and thus precipitate death; but one may not administer potassium chloride, the sole purpose of which is to stop the heart. In short, the decisions of the court and the College were based on the assumption that while passive euthanasia may be permissible in certain circumstances, active euthanasia is never permissible.

This doctrine was explicitly endorsed by the Canadian Medical Association in a policy summary on physician-assisted death published earlier this year.[4] The CMA takes physician-assisted death to include both euthanasia and assistance in suicide, and it states that "Canadian Medical Association members should specifically exclude participation in euthanasia and assisted suicide." But the definition of physician-assisted death is qualified: "Physician-assisted death, as understood here, does not include the withholding or withdrawal of inappropriate, futile or unwanted medical treatment or the provision of compassionate palliative care, even when these practices shorten life."

* * * * *

The case of Dr. de la Rocha and the CMA's recent policy summary on physician-assisted death reveal that James Rachels's well-known article, "Active and Passive Euthanasia," has not influenced the Canadian medical profession's attitudes about euthanasia. In this article, published over 20 years ago in the *New England Journal of Medicine,* Rachels challenges the doctrine that it is sometimes permissible to allow a patient to die by withholding treatment, but never permissible to do something that is intended to kill a patient.[5] He argues that the distinction between killing and letting die is not in itself morally relevant, and so the distinction between active and passive euthanasia is not morally relevant, either. In Rachels's view, we should come to a decision about whether euthanasia is appropriate in a particular case irrespective of the means by which death would be brought about. Then, if we decide that euthanasia is indeed appropriate, we should bring about death by the most humane means, limiting suffering as much as possible. Consequently, in cases in which a dying patient's suffering cannot be adequately relieved with palliative care, active euthanasia would actually be preferable to passive euthanasia, for it brings a quicker end to the suffering.

Rachels's position undoubtedly has an immediate appeal. Once terminally ill patients have decided that their suffering has become unbearable and the time has come for them to die, why should it matter whether death comes from the cessation of treatment or from a lethal injection? Moreover, why insist on the option that in some cases will lead to more suffering? After all, it is the desire to relieve suffering that prompts the decision to bring about death in the first place. But many people will still say that killing *is* worse than letting die. They believe that while it may be morally permissible in certain circumstances to let someone die, it is never morally permissible for one human being to kill another, no matter how good the intention may be. To refute this position, Rachels asks us to consider two hypothetical cases.

In the first case, Mr. Smith stands to gain a large inheritance if his young cousin were to die. Motivated by greed, Smith sneaks into the bathroom while his cousin is taking a bath and drowns her. In the second case, Mr. Jones also stands to gain a large inheritance if *his* young cousin were to die, and he, too, sneaks into the bathroom planning to drown his cousin in the bath. But just as Jones enters the bathroom, the girl slips, hits her head, and falls face down, unconscious, in the water. Jones is poised to force her head back down should it be necessary, but it is not necessary. The girl drowns while Jones does nothing.

Rachels reasons that if there is indeed an *inherent* moral difference between killing and letting die, then this difference must be present *whenever* a case of killing is contrasted with a case of letting die. He then asks whether there is any difference in our moral assessments of the Smith and Jones cases. Surely the answer is no, he argues, for it is ludicrous to say that Jones's behaviour was less reprehensible than Smith's on the grounds that Jones did not actually kill his cousin but "merely" let her die. Rachels concludes, therefore, that the distinction between killing and letting die is not in itself morally relevant.

It must be emphasized that the Smith and Jones cases are not meant to be analogous to cases of active and passive euthanasia. Rather, they constitute a thought experiment designed to prove the premise that there is no inherent moral difference between killing and letting die. For the purposes of the argument, then, it does not matter that in the Smith and Jones cases the motive for bringing about death is malicious, whereas in cases of euthanasia the motive is benevolent.

Philippa Foot believes that Rachels's view is not merely mistaken but "extremely implausible,"[6] and she offers a number of examples in which the distinction between killing and letting die clearly seems to be morally relevant. Here is one of them, slightly embellished. First consider the dilemma of Dr. Brown, who has a limited supply of a drug and six patients who will die without it. The dilemma arises because one patient needs the entire available amount of drug, while the other five need only one fifth of the amount. Should he let one die to save five? Now consider the dilemma of Dr. Green. She has five patients who will all die unless they undergo an organ transplant, but unfortunately the organs they require are not available. It occurs to her, however, that there is a healthy clinical clerk on the ward with all the organs necessary to supply the five patients. Should she kill one to save five?

Both cases involve a choice between life for one person and life for five. The only difference between them is that Brown would have to let one person die to save five, whereas Green would actually have to kill one person to save five. But this difference is critical from a moral point of view, for it is obvious that Brown should choose life for five, whereas Green should choose life for one. We must now face a difficult question: Why does the distinction between killing and letting die seem morally relevant in the Brown and Green cases, but morally *ir*relevant in the Smith and Jones cases? Foot argues that the answer lies in the kinds of rights and duties that are associated with killing and with letting die.

Rights are often divided into two types – negative rights and positive rights. Our negative rights are our rights *not* to be interfered with; these include our right not to be harmed and our right not to have our property taken away. Our positive rights, on the other hand, are our rights to goods and services; *these* include our right to food and our right to medical care. Corresponding to one person's negative and positive rights are other people's negative and positive duties: we have a negative duty not to harm one another and a positive duty to feed the hungry. Notice, then, that killing someone involves the violation of a negative right, the right not to be killed, whereas letting someone die involves the violation of a positive right, the right to be cared for or protected. Or to put it the other way, to kill someone is to fail in a negative duty, the duty not to kill others, whereas to let someone die is to fail in a positive duty, the duty to provide others with care or protection.

Let us now apply this principle to the cases of Smith, Jones, Brown, and Green. The difference between the Smith and Jones cases is that Smith fails in his negative duty not to kill, whereas Jones fails in his positive duty to care for children. The moral significance of this difference is only academic, however, because in the circumstances described we consider it equally bad to fail in either duty. The cases of Brown and Green are more complicated. If Brown lets one die to save five, he fails in his positive duty to provide the one with medical care. If Green kills one to save five, she fails in her negative duty not to kill the one. Here, though, the difference is morally important. In the Brown case, the positive right of the one to receive medical care obviously does not outweigh the positive right of the five to receive medical care; in the Green case, on the other hand, the negative right of the one not to be killed *does* outweigh the positive right of the five to receive medical care. Consequently, it is permissible – indeed, required – for Brown to fail in his positive duty to provide the one with medical care, but impermissible for Green to fail in her negative duty not to kill the one.

Why does a person's negative right not to be killed carry

more weight than a person's – or even five people's – positive right to receive life-saving medical care? The answer, Foot argues, is that "[t]ypically, it takes more to justify an interference than to justify the withholding of goods or services" – that is, it is more difficult to justify violating a negative right than a positive one. In many contexts, of course, it is impossible to justify violating either a negative or a positive right, as in the Smith and Jones cases. But in some contexts, it may be morally permissible or even required to violate a positive right, as in the Brown case, but morally impermissible to violate a negative right, as in the Green case.

* * * * *

I think that Foot's response to Rachels is instructive because it reveals that the context plays a critical role in our moral assessments of killing and letting die. Therefore, we should not blindly apply a single moral analysis of active and passive euthanasia to all contexts in which the issue of euthanasia arises. Instead we should examine each individually and be prepared to come to different conclusions. Another reason for taking a context-by-context approach is that the social consequences of permitting active or passive euthanasia might vary from one context to another. So even if there are a number of contexts in which active or passive euthanasia is morally permissible in itself, the social consequences of permitting the practice might be acceptable in only some of them.

I will now discuss three contexts that involve a decision about euthanasia to illustrate that different contexts require different moral analyses. First, consider patients who rely on extreme medical interventions for their survival, suffer a great deal of pain, and decide that they would like to die. Few would argue that they should be kept alive as long as possible by whatever medical means possible. The question then becomes whether we should allow them to die by withholding treatment, or whether we should kill them with a lethal injection. If we allow them to die, we do not fail in our positive duty to provide medical care, for they have explicitly refused this care. If, on the other hand, they permit us to kill them and we do so, we do not fail in our negative duty not to kill, because they have waived their negative right not to be killed by us. The question must therefore be decided on a different basis, and that should be, as Rachels suggested, which course of action would result in the least suffering. Since a lethal injection would end the suffering immediately, whereas withholding treatment would allow it to continue for a period of time, the former option should be preferred.

Second, consider patients who, though in a great deal of pain and suffering from a condition that is incurable, could continue living for an indefinite period of time without extreme medical interventions. These patients might also decide that they would like to die. Here passive euthanasia is not an option, for there is no treatment to withhold. The choices before us are active euthanasia or no euthanasia at all. One might argue, as in the previous context, that active euthanasia would be permissible because the patients would be waiving their negative right not to be killed. We must also consider, however, the social consequences of permitting active euthanasia in this context.

A strong argument could be made that overzealous heirs or even overzealous doctors might pressure these patients to consent to being killed. Patients who would not otherwise consent might do so if they felt that they were a burden on their families. If active euthanasia became a more common choice, there might be less impetus to do research in palliative care. And no doubt there could be other unacceptable social consequences. To determine whether such consequences would in fact ensue, we would have to call on experts from a variety of fields, including medicine, nursing, social work, psychology, and sociology. We would also want to examine the many studies on the impact of euthanasia in the Netherlands, where the practice, though not technically legal, is tolerated as long as doctors follow established guidelines.[7] Nonetheless, I suspect that active euthanasia should be prohibited in this second context, for it seems likely that permitting it would indeed have unacceptable social consequences.

Some might argue that patients might feel pressured to consent to active euthanasia in the first context as well as in the second, and so we should prohibit the practice in both contexts. But while it is true that in the first context patients might feel pressured to consent to active euthanasia, these patients might also feel pressured to consent to passive euthanasia. Therefore, proponents of this argument would have to accept the conclusion, opposed by most, that in the first context we should also prohibit passive euthanasia. This is reason enough, I think, to reject the argument. Notice that prohibiting active euthanasia in the *second* context is not open to the same objection, because passive euthanasia is not an option there.

In both contexts just discussed, the patients are assumed to be competent and have expressed an explicit wish to die. Euthanasia is therefore said to be "voluntary." Sometimes, however, it is impossible to know patients' wishes with regard to euthanasia – for example, if they are physically unable to communicate their wishes, if they are mentally incompetent, or if they are newborns. Here euthanasia is said to be "nonvoluntary." Nonvoluntary euthanasia introduces new problems, and far fewer people are in favour of it. Even in the Netherlands, nonvoluntary euthanasia is prohibited. Nonetheless, let us examine one context in which the question of nonvoluntary euthanasia would arise.

Consider patients who are in a persistent vegetative state and have never expressed, either orally or in a document such as a "living will," what they would want to happen to them in such circumstances. A number of reasons might be given for wanting to bring about the death of such patients – for example, we might think that they have such a poor quality of life that they would be "better off dead," or we

might reason that the resources required to sustain them should be used for other medical or social purposes. In addition, family members might want the patient to die, perhaps because they think that that is what the patient would have wanted, or perhaps because they feel that they are no longer able to cope emotionally with the situation. In considering active euthanasia, we must decide whether such reasons outweigh the patient's negative right not to be killed. In considering passive euthanasia, on the other hand, we must decide whether they outweigh the patient's positive right to receive medical care.

Although it may be tempting, we cannot simply assume that these patients would waive their negative right not to be killed or their positive right to receive medical care. We might appeal, however, to Foot's assertion that it takes more to justify the violation of a negative right than the violation of a positive one. Given this, we could argue that while the reasons in favour of bringing about death do not justify the violation of the negative right not to be killed, they might justify the violation of the positive right to receive medical care. Therefore, while active euthanasia would be ruled out, passive euthanasia might be morally permissible. The question remains, however, whether the reasons in favour of bringing about death would ever, in fact, outweigh the patient's positive right to receive medical care. This is a difficult question, and I will not attempt to answer it here. It is also difficult to anticipate the social consequences of permitting passive euthanasia in this context.

In each of the three contexts discussed, not only the analysis but also the conclusion was different. In the first context – where patients who rely on extreme medical interventions for their survival ask to die because they can no longer tolerate their suffering – I concluded that euthanasia is morally permissible, and that active euthanasia should be preferred over passive. In the second context – where patients ask to die for the same reason even though they could live for an indefinite period of time without extreme medical interventions – passive euthanasia is not an option in the first place, and I reasoned that while active euthanasia is morally permissible in itself, it should probably be prohibited because of the likely social consequences of permitting it. In the third context – where patients are in a persistent vegetative state and have never expressed what they would want to happen to them in such circumstances – I concluded that while active euthanasia is ruled out, passive euthanasia might sometimes be permissible.

I would like to emphasize that my aim has not been to provide an exhaustive list of contexts in which the issue of euthanasia would arise. Rather, my aim has been to show that it is impossible to discuss whether passive euthanasia is preferable to active, or whether euthanasia should be permitted at all, without considering the particular context, for the moral implications will vary from one context to another. Too often, the subject of euthanasia is approached with the attitude that one must come to universal conclusions. Although people may have different intuitions concerning different cases, they try to resist these intuitions, believing that they must be consistent in their views. But this is a foolish consistency. The moral problems associated with euthanasia are complex; there is no reason to expect the solutions to be simple.

In adopting the policy that doctors may participate in passive but not active euthanasia, the CMA has failed to recognize (1) that the moral implications of active and passive euthanasia are different in different contexts, and (2) that in at least one context, active euthanasia should be permitted and even preferred over passive euthanasia. Instead of developing a single policy on active and passive euthanasia, policy makers should identify all the contexts in which the issue of euthanasia would arise, and for each one develop a separate guideline that is based on a separate moral analysis. Furthermore, the analysis should address not only whether, in a given context, active and passive euthanasia are morally permissible in themselves, but also the social consequences of actually permitting the practices in that context. Until guidelines developed in this way are in place, doctors faced with a decision about euthanasia, as Dr. de la Rocha was four years ago, will be under pressure to follow a policy that is sometimes morally inappropriate.

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Prospects of Arresting the Progression of Age-Related Pathophysiologic Processes by Dehydroepiandrosterone

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Abstract

The aging process is accompanied by an increase in body fat, decreased protein synthesis, and decreased lean body and bone mass. These changes, which predispose to pathologic conditions are associated with steady and progressive declines in the levels of dehydroepiandrosterone (DHEA) and its sulfate ester metabolite, dehydroepiandrosterone-sulfate (DHEAS). The role of DHEA and its sulfate was first determined to be an intermediate in sex steroid synthesis. More recent scientific evidence has shown that DHEA is involved in many biologic actions, implicating a connection between declining DHEA levels with age and increased incidence of a variety of pathophysiologic conditions in the elderly, such as cardiovascular disease, atherosclerosis, cancer, osteoporosis and Alzheimer's disease. There are many unanswered questions about the role of DHEA with respect to the neuro-immuno-endocrinological network and there still remains the question of whether this age-related DHEA decline is simply due to organ-specific failure at the level of the adrenals or gonadal organs. Nevertheless, DHEA shows a gradual decline reaching its lowest level with advanced aging. As very few compounds in our bodies show such a phenomenon, it is possible that DHEA affects many physiological processes occurring with age. It has, thus, been postulated that replacement therapy of DHEA may prevent and perhaps even improve certain conditions commonly occurring in the aged. DHEA may very well be the forever searched *"fountain of youth"*.

Résumé

Le processus de vieillissement s'accompagne d'une augmentation de la graisse corporelle et d'une baisse de synthèse protéique, de masse osseuse et de la 'masse maigre'. Ces changements qui prédisposent à de conditions pathologiques s'accompagnent d'un abaissement progressif du déhydroépiandrostérone (DHEA) et de son métabolite, le sulfate de déhydroépiandrostérone (DHEAS). Le premier rôle démontré du DHEA et de son sulfate fut celui d'intermédiaire dans la synthèse des stéroïdes sexuels. Plus récemment, les preuves scientifiques démontrent plusieurs autres actions biologiques du DHEA, impliquant une relation entre le déclin pro-

gressif avec l'âge du taux de DHEA et l'incidence accru d'une variété de conditions pathologiques chez la personne âgée, dont la maladie cardio-vasculaire, l'athérosclérose, le cancer et la maladie d'Alzheimer. On s'interroge sur le rôle du DHEA dans le réseau neuro-immuno-endocrinien et sur la possibilité que son déclin pourrait simplement être dû à l'affaiblissement progressif de la fonction des surrénales ou des gonades. Il n'en demeure pas moins que le taux de DHEA décroit à mesure que l'on avance en âge atteignant son plus bas niveau à l'âge le plus avancé. Comme peu de 'composés' de notre corps démontrent un tel phénomène, il est possible que le DHEA affecte plusieurs processus physiologiques survenant avec l'âge. L'hypothèse qu'une thérapie de remplacement du DHEA pourrait prévenir on même améliorer certaines conditions survenant communément chez la personne âgée a été suggérée. Le DHEA deviendrait en quelques sorte une *" fontaine de jouvence"*.

Dehydroepiandrosterone (DHEA)

For many years it has been known that dehydroepiandrosterone (DHEA) and its metabolite, dehydroepiandrosterone-sulfate (DHEAS), are secreted by the adrenal cortex in abundant quantities, but their definitive biological accord has yet to be ascribed[1]. These steroids are biotransformed into active estrogens and androgens, as outlined in Figure 1. It does not, however, appear that they have a primary role as sex hormone precursors in the human body[2]. Studies have shown that administration of DHEA, but not DHEAS, to laboratory animals gives rise to an array of biological effects[3, 4], although the mechanism of action can only be speculated and, thus, remains unclear.

Circulating levels of DHEA and DHEAS are higher at birth than the upper limits reported in adults[5, 6] and then fall markedly until adrenarche[5], when there is a marked increase of the steroids in both sexes[6, 7]. The concentrations of DHEA and DHEAS peak during the third decade of life and then progressively decline to low levels at senescence[8]. It has been postulated that these fluctuations in adrenal secretion of androgen throughout the lifespan, with the absence of any significant changes in cortisol secretion, are due to a decrease in 17,20-desmolase activity in the adrenal cortex[9-12]. However, the significance in the fluctuation of DHEA and DHEAS production from young adulthood to old age is still not clear.

Dehydroepiandrosterone has been implicated in the modulation of such pathophysiologic events as diabetes, obesity, carcinogenesis, neoplastic growth, neurite outgrowth, viral and bacterial infections, stress, pregnancy, hypertension, collagen and epidermal integrity, fatigue, depression, memory and immune responses[2]. Here, we will discuss many of these proposed effects of DHEA with reference to the original studies, in hope of providing a clear perspective on the possible role of DDHEA and hence predict the benefits of therapeutically administered DHEA.

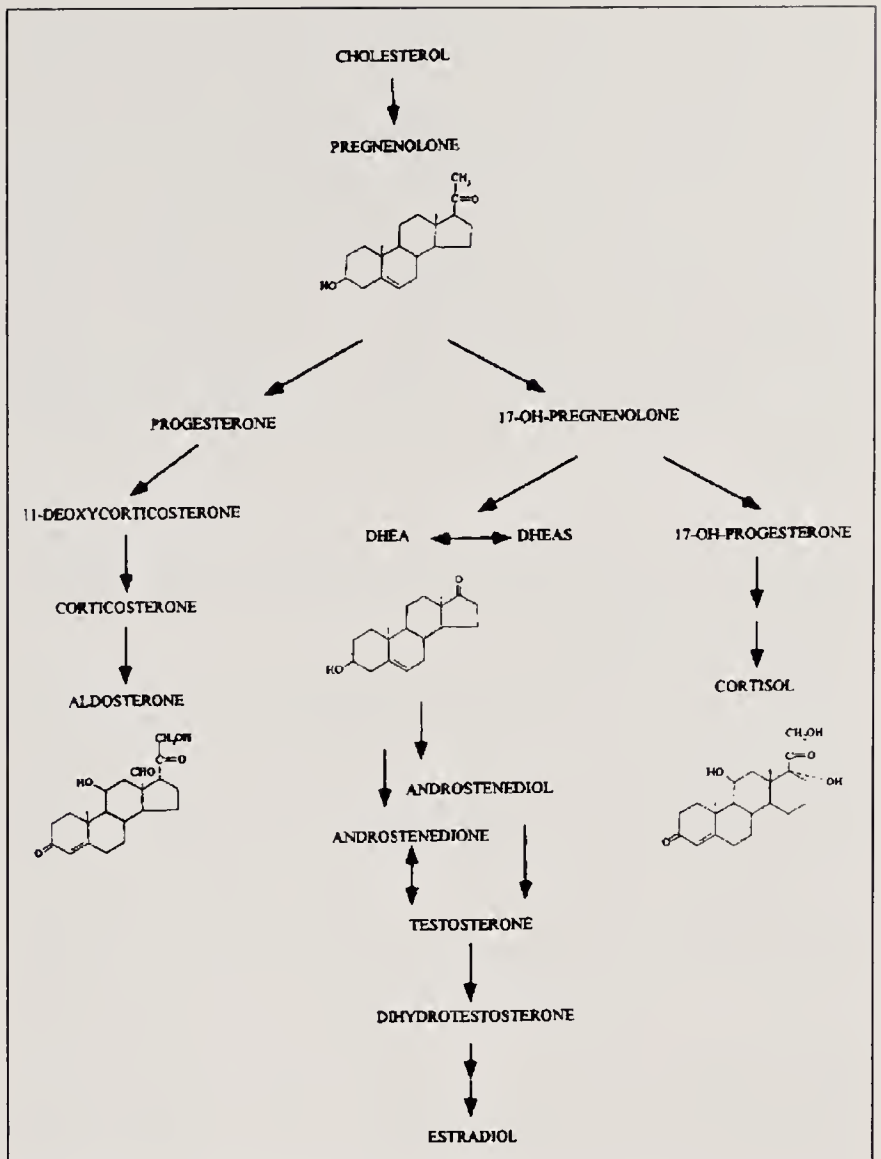

Figure 1: *Pathway of steroid synthesis in the adrenal cortex.*

Cardiovascular Effects of DHEA

The proposed association between DHEA and DHEAS and cardiovascular disease was studied in 1135 men and 567 women from Rancho Bernardo, California between 1972 and 1974[13]. In this study the plasma levels of DHEAS were measured in men aged 30 to 79 and in women aged 50 to 79. DHEAS levels decreased with each decade, a unique phenomen not observed with other sex hormones after age 50 measured in this study, including estrone, estradiol, testosterone and androstenedione. In this study, DHEAS levels were inversely associated with cardiovascular disease in men. However, in women it was positively associated, suggesting that unlike in men a high level of DHEAS is not predictive of a reduced risk of heart disease in women. Similarly, sex differences have been reported with angiography in which DHEAS levels were inversely correlated to coronary atherosclerosis in men but not in women[14]. It is thought that women have less heart disease than men due to the presence of higher estrogen levels, and that estrogen therapy reduces the risk of cardiovascular disease in postmenopausal women[15]. Conversely, it has been documented that estrogen thera-

py in men leads to an increased risk of vascular disease[15]. The question that arises is why different sexes repond differently to hormone therapy? Perhaps it is the unopposed androgens produced by the adrenals as much as the decrease in estrogen in menopause that cancels the advantage females have over males with respect to the development of cardiovascular disease. Future studies addressing the potential actions of DHEAS may aid in elucidating this unexplained increased risk of premature heart disease in men as compared to premenopausal women.

Effects of DHEA on Cholesterol and Lipoproteins

Elevated levels of total serum cholesterol and in particular low density lipoproteins (LDL) have been implicated as high risk factors for the development of atherosclerosis in human[16]. The development of this condition may be a result of complex interactions among nutritional, environmental, hormonal, and genetic factors. Atherosclerosis was proposed to be a 'response to injury' associated with damaged endothelium, proliferating smooth muscle cells, and LDL-laden macrophages, or foam cells, in the intima of the vessel[17-23]. This progressive proliferation is influenced by increased levels of cholesterol, especially LDL, adherence of platelets and macrophages, the release of chemoattractants and growth factors, and cell proliferative ability. DHEA may in some way modify many of these associated factors of the atherogenic process.

The mechanism(s) of DHEA's anti-atherogenic and anti-obesity effects have been postulated, but not proven. A decrease in DHEA and DHEAS concentrations increase lipogenesis because DHEA is a non-competitive inhibitor of mammalian glucose-6-phosphate dehydrogenase (G-6-PD), the rate-limiting enzyme of the phosphate Pentose shunt and major source of extra-mitochondrial NADPH[24]. NADPH is necessary for the reductive synthesis of fatty acids and cholesterol[25]. The rate-limiting step in the biosynthesis of cholesterol is hydroxymethylglutaryl-CoA (HMGCoA) reductase, which converts HMGCoA to mevalonate, requires NADPH in order to proceed[26]. NADPH is also required for the production of D-ribose-5-phosphate, which is used in nucleotide biosynthesis through the intermediate 5-phosphoribosyl-1-pyrophosphate. Purine ribonucleotide as well as thymidylate synthesis rely upon tetrahydrofolic acid which requires NADPH for its function. The enzymatic formation of deoxyribonucleotide diphosphates by ribonucleotide reductase is also dependent on NADPH. The atherogenic process is thought of as a proliferative process[28], much like the neoplastic process requiring DNA synthesis, which is inhibited by DHEA[23, 27].

The effect of DHEA on the activity of key regulatory enzymes in liver was investigated[28] and there appeared to be a rapid decrease in pyruvate kinase activity and an increase in malic enzyme activity. These changes reflect a decreased rate of glycolysis accompanied by an increased rate of gluconeogenesis, similar to those observed with fasting. This low pyruvate kinase activity may therefore be suggestive of a shift from fat metabolism to carbohydrate metabolism in liver. The authors of this investigation further postulate an increased rate of fatty acid utilization by either beta-oxidation or peroxisomal oxidation[28].

Some other plausible mechanisms by which DHEA could modulate the atherogenic process are by affecting the adherence of platelets and macrophages, the release of chemoattractants and growth factors, and the formaton of superoxide free radicals[22]. It has been reported that DHEA blocks superoxide anion production[29] and that DHEA may reduce the pool of arachidonic acid which is metabolized to various eicosanoids through both the 5- and the 12-lipoxygenase pathways. These pathways generate different hydroxy-fatty acids and leukotrienes, and through the cyclooxygenase pathway specific prostaglandins, prostacyclins and thromboxanes are formed[29].

In humans, administration of adrenal androgens produced changes in cholesterol and lipoprotein levels. It was prevously reported[30] that androsterone administered to hypothyroid patients for 3 to 34 days decreased serum cholesterol by about 22%. Studies utilizing DHEA administration[31-35] also reported diminuating cholesterol levels.

Another interesting study, in which DHEA was administered to healthy men, reported reduced serum low density lipoprotein (LDL) levels as well as body fat without any change in insulin sensitivity[36]. This study predicted a 14% reduction in risk for developing cardiovascular disease upon administration of 1600 mg of DHEA for 28 days. Other hormones measured, including testosterone, sex hormone-binding globulin, estradiol, and estrone, showed no change. The change in lipid profile observed with DHEA is quite different from that observed with other androgens which characteristically lower high density lipoprotein (HDL) levels and increase LDL levels. Hence, DHEA effects are more in line with estrogen-like effects[37].

Central Nervous System Effects of DHEA and its Sulfate

When it is desired to ameliorate nervous system function, such as by increasing the rate of repair of neurons damaged by disease, injury or aging, or enhancing learning and memory, then one must facilitate adaptive coupling among relevant functional processes by relieving rate-limiting parameters in mutually shaping interactions among intracellular, intercellular and extracellular processes of the system[38]. Administration of DHEA and DHEAS was shown to enhance survival of neurons in explants of fetal mouse brain in culture[39, 40]. Increased

neuritic outgrowth processes as well as the formation of connections between them were observed[39, 40]. DHEA and DHEAS also enhanced memory in both young and old mice[39, 41, 42]. It has previously been suggested that DHEA and DHEAS exert their effects by facilitating of the operation of the cyclic GMP (cGMP) cycle, perhaps by activating guanylyl cyclase, which could be the rate-limiting step in the cycle itself[38]. This leads to the possibility that DHEA and DHEAS may also protect nervous tissue *in vivo* against damage and facilitate regeneration when necessary. Consistent with this hypothesis[38], serum levels of DHEAS were on average 48% lower in patients diagnosed with Alzheimer's disease as compared to age-matched controls. Perhaps the administration of DHEA and DHEAS to these patients may improve their condition by releasing diverse aspects of metabolic activity necessary for intracellar and extracellular communication to occur[43].

DHEA and Cancer

The mechanism(s) of cancer prevention by DHEA has not yet been unravelled, but many theories have been proposed. One laboratory reports DHEA's anti-carcinogenic effects to be attributed by its inhibition of G-6-PD with a block of carcinogen synthesis or activation and/or block of ribose-5-PO_4 and impeded synthesis of DNA. Investigations have revealed a proportional relationship between G-6-PD activity and hepatocyte ploidy, using mononuclear diploid stem cells having elevated G-6-PD activity[44]. Another study describe DHEA acting through depletion of NADPH, thus, inhibiting free radical formation[3]. Further studies in liver show these anti-carcinogenic effects of DHEA to be mediated by a differentiating effect on pre-neoplastic nodules and that this is associated with peroxisome and catalase induction[45-47].

In search for a uniform theory for DHEA's anti-carcinogenic properties, an alternative hypothesis has been brought forth. Perhaps the slowing of hepatic cellular proliferation induced by DHEA could produce a cytokine activated differentiation, or conversely a cytokine induced cell stasis, or simply cell death[48].

DHEA is also a sensitive indicator for patients at risk for breast cancer[49-51]. It has been shown that individuals with subnormal plasma and urinary excretion levels of DHEA and DHEAS in those with high risk for breast cancer and poor prognosis[52]. However, patients with high DHEA plasma ratios respond well to endocrine therapy. Therefore, measuring the levels of DHEA may be a way to predict therapeutic response in metastatic disease[53].

In vitro, breast cancer cells can convert DHEA and DHEAS to hydroxy-DHEA and androstenediol. It has been suggested that androstenediol competes with estrogen for its receptors, probably inhibiting its proliferative action[54,55]. Hence, the conversion of DHEA to androstenediol might play a role in breast cancer.

Studies on DHEA Replacement Therapy

In humans, the aging process is accompanied by a progressive decline in the secretion of DHEA and its sulfate, and of GH-insulin-like growth factor-I (GH-IGF-I). While the functional relationship of declining GH-IGF-1 and catabolism is recognized, the biological role of DHEA in aging remains unclear. A recent study[56] addresses this issue by administering a physiological replacement dose of 50 mg of DHEA at bedtime to 13 men and 17 women between the ages of 40 and 70. This dose of DHEA restored DHEA and DHEAS to levels normally found in humans in the second decade of life[8], which were maintained for 3 months. This study[56] showed that DHEA produced an approximate 10% elevation in serum IGF-I levels and an approximate decline of 19% in IGF-binding protein-1 (IGFBP-1), leading to an elevation of IGF-I/IGFBP-I ratio by 50% in both women and men. These changes were accompanied by an increase in perceived 'well-being', both physiological and psychological, for 67% of the men and 84% of the women in the study. The mechanism underlying improved 'well-being' is not yet understood, but there is a possibility that DHEA has a central effect[57].

This study[56] did not report any negative side effects resulting from administration of 50 mg DHEA for six months. Such positive results warrant future trials to determine the biological consequences of elevated bioavailable IGF-I in healthy aging people.

Concluding Remarks

DHEA and DHEAS have been widely studied in clinical trials and to date no adverse effects have been reported. Currently, much clinical interest is focused on the use of DHEA in the prevention and treatment of cancer and atherosclerosis. However, due to DHEA's wide spectrum of biological activity, it has much broader potential in clinical medicine. As stated by Regelson and co-workers[48], "... DHEA, as a precursor steroid, can be likened to a 'mother steroid' whose idealized role is to provide useful progeny and to help these varied steroids provide for a state dependent or hormone modulating existence". By tapping into the mechanisms of action of DHEA in the human body, we can exploit these actions in order to improve certain diseases and perhaps even arrest the progression of some age-induced diseases.

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Inferior Vena Cava Filters - What's New in 1995

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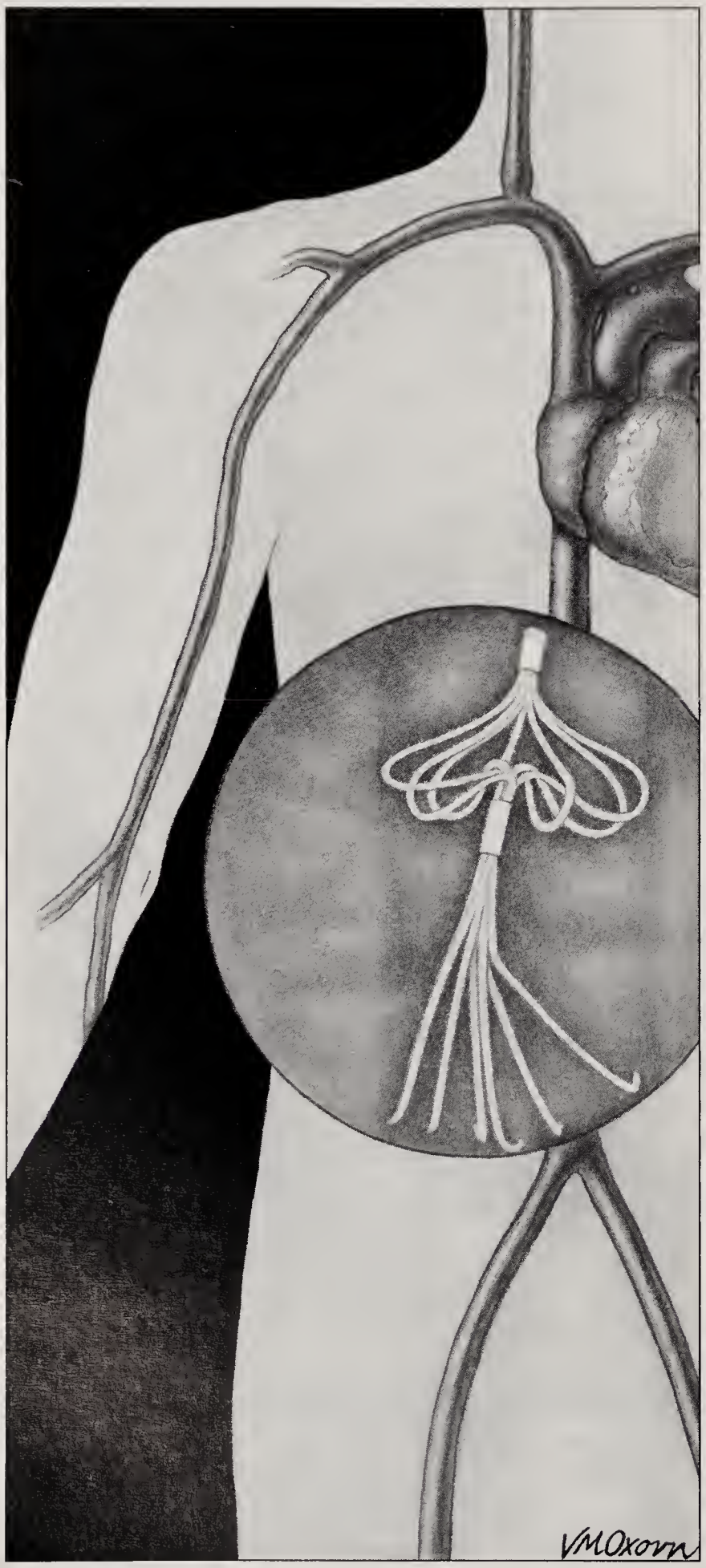

VALERIE OXORN

Abstract

The Simon Nitinol (SN) inferior vena cava (IVC) filter was recently released in Canada for the treatment of patients at risk for pulmonary embolic disease. We report 14 patients who received SN IVC filters based on the following clinical indications: contraindication to anticoagulation (71%); failure of anticoagulation (8%); prophylactic placement (14%); and complication of anticoagulation (8%). Traditional approaches through the femoral and jugular veins are used with other filter designs, and may be associated with increased complications. In this study, 58% of patients had the SN filter inserted through the basilic vein. No filter related complications, recurrent pulmonary embolism, or access site complications were noted on follow-up from 3-196 days. The advantages that the SN filter provides over other filter designs are increased flexibility, alternative approaches for insertion, and decreased complications.

Introduction

Pulmonary embolic disease (PE) is a common, life threatening condition. Untreated, the associated mortality rate is 26%, however with appropriate therapy, mortality is reduced to 2.5%.[1,2] Pulmonary embolism commonly occurs in the setting of lower extremity deep venous thrombosis (DVT), which may be asymptomatic. Standard medical therapy includes anticoagulation, however in certain clinical situations, interruption of the inferior vena cava (IVC) may be necessary. IVC filters are indicated when there is a contraindication to anticoagulation, failure of adequate anticoagulation, or a complication while on anticoagulation therapy. Filters may also be placed prophylactically in high risk patients, or in patients with severe cor pulmonale and pulmonary hypertension. Contraindications to percutaneous IVC filter insertion are unusual, and would most commonly include an uncooperative patient.

The first IVC filter used in clinical practice was the Mobin-Uddin filter which was introduced in 1967. This filter had a 70% caval occlusion rate.[3,4] The stainless steel Kimray Greenfield filter became available in 1973, and was the most widely utilized filter until the last several years. The large, 24 French (F) introducer system for this

Figure 1: *Simon Nitinol filter.*

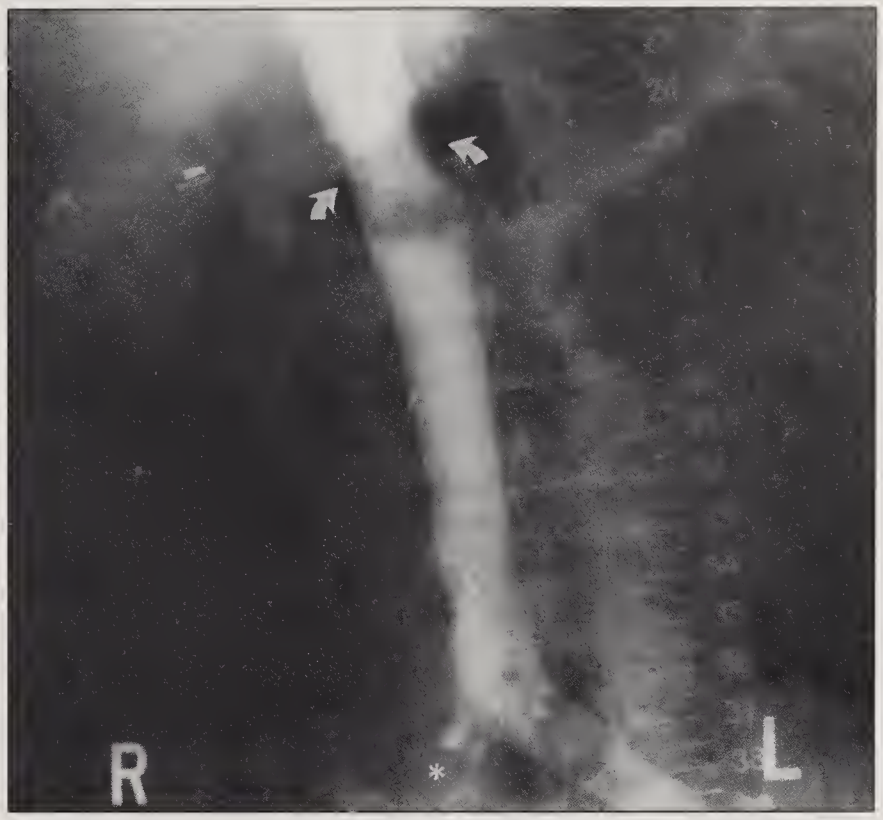

Figure 2: *Venacavagram via the jugular approach shows a thrombus in the right common iliac vein (asterisk) and inflow defects at the level of the renal veins (arrows).*

filter resulted in intraoperative placement via surgical cutdown, however these filters were also inserted percutaneously in Radiology departments.[3,4,5]

Recent developments in filter design include a smaller introducer size (9-12F), the use of different materials (Titanium, Nitinol), and modification of filter design (side struts to improve stability, and modifications to increase flexibility).[5] Other currently available filters are the Birds Nest filter and the Vena Tech filter. The goal of our study was to evaluate the SN filter which has been recently released in Canada. The unique composition, flexibility, and small introducer should result in simpler insertion, and a lower complication rate in comparison with other currently available filters.

Methods

This study was performed at a quaternary care teaching hospital in Toronto. All patients referred for IVC filter placement were included in this study which began at the time the SN filter (Figure 1) was made available to our institution in February 1995, and extended to August 1995. A total of 14 patients received IVC filters during this period.

Indications for IVC filter placement: contraindication to anticoagulation in 10 (71%), failure of anticoagulation in 1 (8%), prophylaxis in 2 (14%), and complication of anticoagulation in 1 (8%). No patients being referred for IVC filter patient were excluded from this trial.

The procedures were performed using local anesthesia, with intravenous Sublimaze (Fentanyl) and Versed (Midazolam) for sedation. Using ultrasound guidance, the desired vein was accessed with an 18 gauge needle, and following successful venous puncture, a guidewire was advanced, and digital subtraction cavography was performed with a 5F pigtail catheter. The location of the renal veins was noted and a metallic marker was placed overlying this level on the patient's abdomen. The size of the IVC was measured, and the IVC was evaluated for thrombosis, and accessory vessels (Figure 2). Following placement of the 9F introducer, the filter was deployed as per the manufacturer's instructions. Follow-up cavography was performed to confirm correct filter placement. Following placement, the sheath was removed, and hemostasis was obtained. Follow-up radiographs were

Figure 3: *Abdominal radiograph demonstrates Simon Nitinol filter in position. Note incidental calcified uterine leiomyoma (fibroid).*

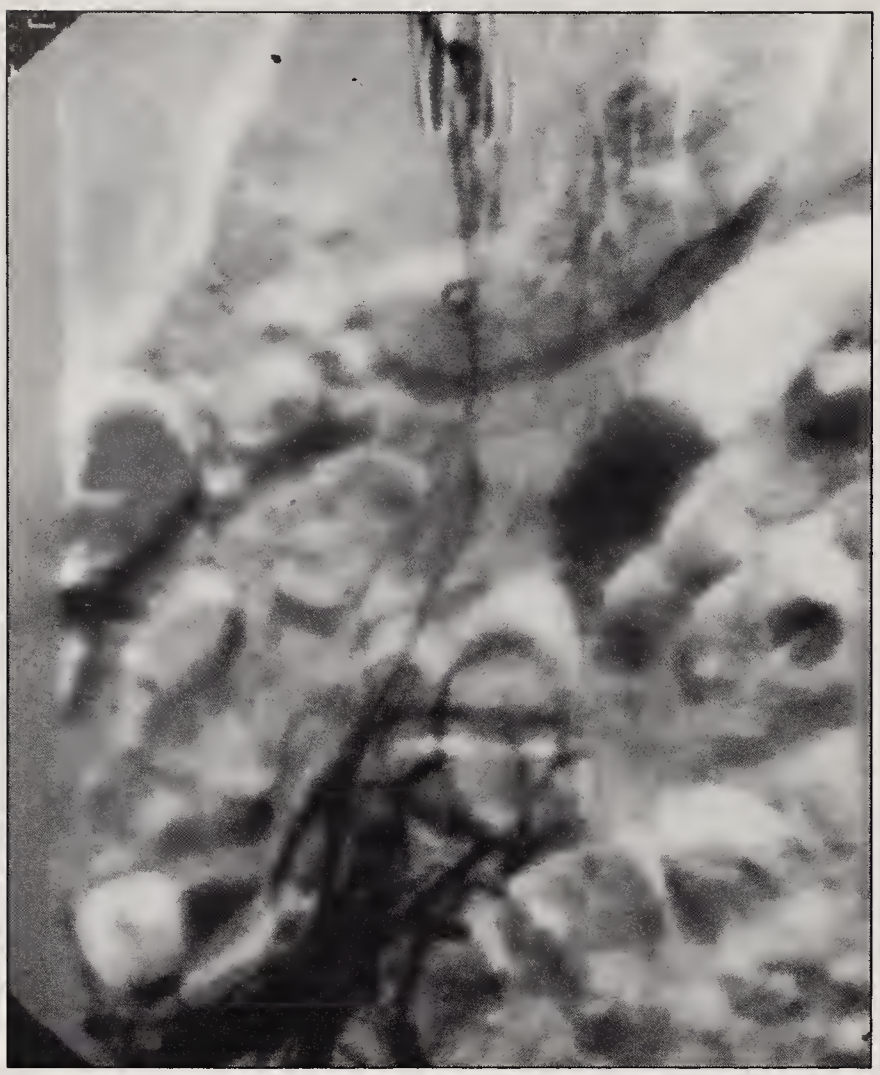

Figure 4a: *Venogram demonstrating IVC thrombosis secondary to an IVC filter.*

Figure 4b: *Post thrombolysis venogram demonstrates clot lysis and reconstitution of flow.*

obtained immediately after filter deployment (Figure 3) and at 1 and 7 days to assess for filter migration.

Results

All insertions were technically successful. Venous access sites in our patients were: basilic vein in 8 (58%), right common femoral vein in 3 patients (21%), and right internal jugular vein in 3 patients (21%).

Patient follow-up ranged from 3 to 196 days. There were no filter related complications, recurrent PE, or access site complications were noted. Four patients died of unrelated causes.

Discussion

IVC filters have undergone numerous modifications since their development in the late 1960's. The desirable characteristics of an ideal IVC filter include: ability to be percutaneously inserted, small introducer size for low insertion related complications, high thrombus trapping ability, preserve caval patency, and biocompatibility. The SN filter is constructed of an alloy composed of nickel and titanium. It is stable in a magnetic field, thus patients with these filters may undergo Magnetic Resonance Imaging (MRI). This material has a unique thermal memory which allows it to be reshaped into a form which can be loaded into a 9F introducer, when cooled to 4-10^{o}C. When exposed to blood at body temperature, the alloy returns to its predetermined shape. This filter can fit through a smaller introducer system than any other currently available filter, and does not require a rigid introducer, resulting in increased flexibility allowing it to negotiate tortuous vessels.[6] An advantage of the smaller introducer size is a decreased incidence of insertion site thrombosis[7], and its flexibility allows it to be inserted through the antecubital veins, or from a contralateral approach which is contraindicated in several other filter designs.

Common IVC filter insertion sites include the femoral and jugular veins. The Bird's Nest and Vena Tech filters are introduced using these 2 routes only. Following access of the femoral vein with a large introducer system, the patient must be confined to bedrest for 4-6 hours to avoid a hematoma. As well, insertion via this site may be more prone to secondary infection. The femoral veins cannot be used when there is underlying ileofemoral thrombus present. A common alternate access site is the right internal jugular vein with the procedural related risk of pneumothorax and carotid arterial puncture. Many patients prefer the access site in the arm, which is a common site for venous puncture. Puncture related complications are low in this region, particularly when ultrasound is utilized. It is easy to achieve and maintain hemostasis at this site as well.

Complications of IVC filters are similar for all types, and include recurrent PE (0.5-7%), IVC thrombosis (0-30%) (Figure 4), insertion site thrombosis (2-41%), and

filter migration (0-36%).[1,7,8] IVC perforation by a filter strut is common with the Greenfield design, but is seldom symptomatic. However, this may result in a small bowel volvulus.[4,9] In our study there were no complications, however the number of patients was small and follow-up was relatively short.

Conclusion

IVC filters are commonly used devices in the treatment and prevention of pulmonary embolic disease. Although there are numerous devices available, the recently released Simon Nitinol filter has the advantage of the smallest currently available introducer system, and the most flexibility, allowing for insertion via an antecubital vein.

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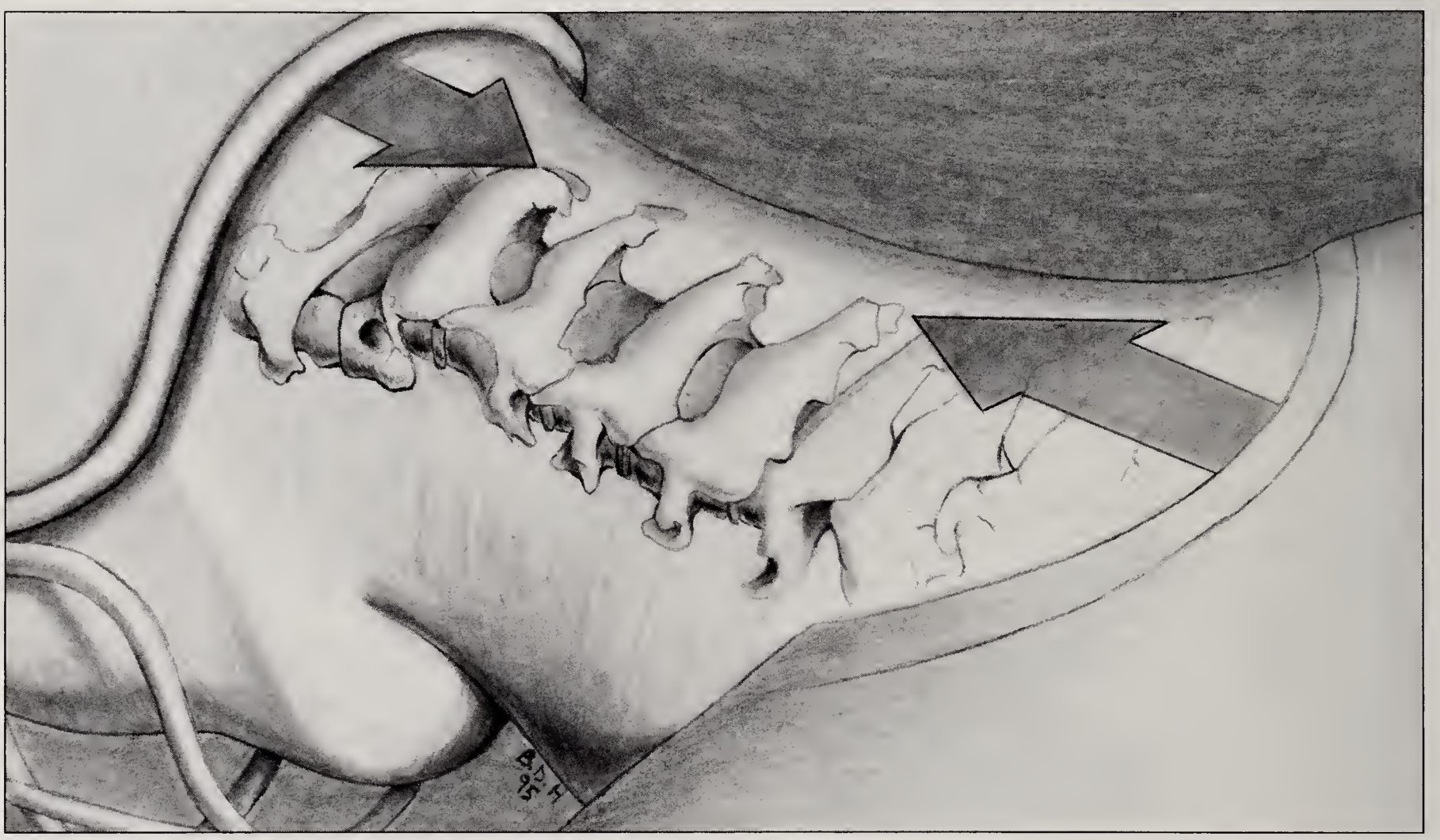
Football Cervical Spine Injuries

Vikas Tuli, B.Sc.Honours, (9T6), and Michael Ford, M.D., F.R.C.S.(C)*

* Surgical Staff, Orthopaedic and Arthritic Hospital, Lecturer, Department of Surgery, University of Toronto



BRYCE HOUGH

Abstract

Football is one of the leading causes of recreational cervical spine injuries. Studies of the pathomechanics of injury and the subsequent implementation of safety measures, such as the "no spearing" rule, have led to a decline in the number of football-related cervical spine injuries. An understanding of football cervical spine trauma requires a reliable accident database such as the American National Football Head and Neck Injury Registry to report incidence and trends of neurotrauma. Pathomechanic studies show that axial loading of the cervical spine is the principle mechanism of injury in football. Axial loading mechanisms such as "spear tackling" cause neurapraxia of the cervical cord with transient quadriplegia and spear tackler's spine, two recently accepted clinical entities.

Introduction

A sports doctor once said, "There is probably no better experimental or research laboratory for human trauma in the world than the football field..."[1] One need only watch a few plays of amateur football to realize the intensity and shocking potential for neurotrauma. Fortunately, the incidence of severe football and other sports-related cervical spine trauma is infrequent[2]. Football injury is the topic of this paper not because it ranks amongst the highest of cervical spine-inflicting athletics, but because of the vast football studies already undertaken. It is a domain largely influenced and dictated by orthopaedic surgeon, University of Pennsylvania's Joseph Torg.

Epidemiologic studies have consistently associated cervical neurotrauma with diving, hockey, gymnastics and football.[2,3] Canadian statistics show the aforementioned

Table 1. Comparison of the occurrence of injuries reported by the National Football Head and Neck Injury Registry. (From Reference 4, with permission.)

	INTRACRANIAL HEMORRHAGES	INTRACRANIAL DEATHS	CERVICAL SPINE FRACTURES/SUBLUXATIONS DISLOCATIONS	PERMANENT CERVICAL QUADRI-PLEGICS
Schneider (1959 to 1963)	139 (3.39/100,000)*	65 (1.58/100,000)	56 (1.36/100,000)	30 (0.73/100,000)
H/N Registry (1971 to 1975)	72 (1.15/100,000)	58 (0.92/100,000)	259 (4.14/100,000)	99 (1.58/100,000)

*annual injury rate per 100 000 participants is in parentheses

sports account for roughly 65% of all recreational spinal cord trauma.[3] The study of the pathomechanics of these sports-related cervical injuries is a necessary and achievable goal. This is an undertaking that has already caused implementation of successful safety measures at both the professional and amateur levels.

The cervical spine and its brachial plexus are prone to injuries which vary from simple sprains to fracture-dislocations with quadriplegia. Fortunately, the frequency of these injuries are inversely proportional to their severity.[2] Minor reversible injuries are seen quite frequently on football fields. Classical teaching regarding cervical spine injuries in sports concentrated on hyperflexion mechanisms.[1] The athlete was thought to be an unsuspecting victim of a force that causes the neck to hyperflex (ie. falling on back of head). The latest clinical reports however refute this[4] and adopt cervical spine axial loading as the principle mechanism of injury. The athlete is subject to a significant axial load that compresses the cervical spine between the head and body.[5] This paper will explain the axial loading mechanism and relate its severe sequelae.

Football History

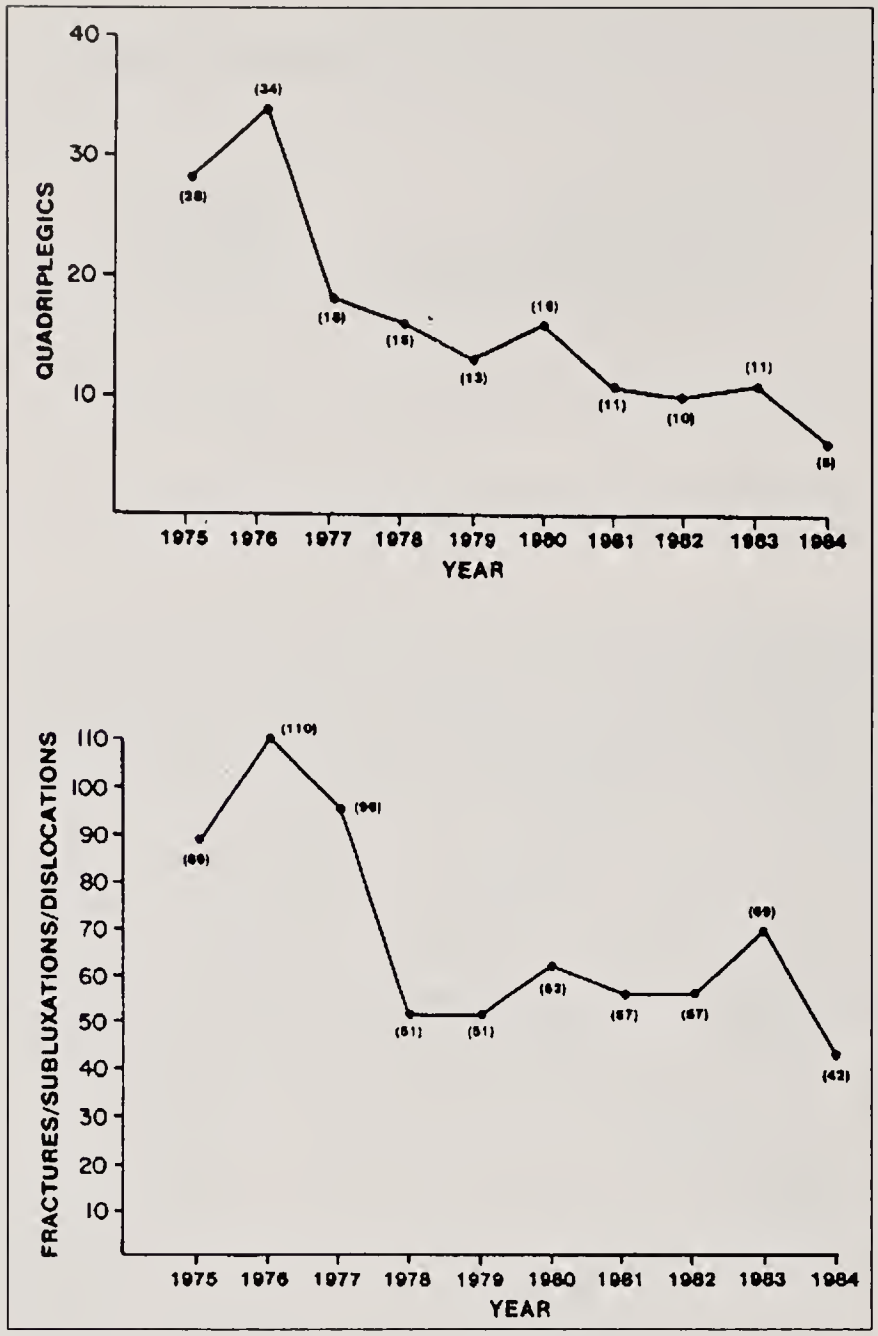
Football cervical spine injuries have been studied and documented in great detail. The progress that has been made in the understanding of the pathogenesis of football cervical injuries is enormous. Part of this success is due to the establishment of the National Football Head and Neck Injury Registry, in 1975. This organization has compiled statistics which show the trends in cervical spine injuries of all levels of American football.

Head and neck injuries in American football have taken a dramatic change since the 1960s. When comparing the 4 year time frames 1959-1963 and 1971-1975, one sees a decrease in intracranial haemorrhages and intracranial deaths. In the same time periods, however, the number of cervical spine fractures and/or dislocations and permanent quadriplegics has increased (Table 1).[1,6] This is due to the improved helmet-face mask unit that developed in the late 1960s. This helmet provided for better intracranial protection but on the down side, allowed the head to be used as the primary point of contact in blocking and tackling.[4] This introduces the phenomena of "spearing".

Between 1971-1975, 52% of the permanent football-induced quadriplegics were caused by "spearing injuries".[4] The injured player makes contact with his opponent with the top of his helmet. This discovery prompted the American high schools and the National College Athletic Association (NCAA) to implement preventative actions. The NCAA came forth with the following rules in the 1976 season: (1) No player shall intentionally strike a runner with the crown or top of the helmet; (2) spearing is the deliberate use of the helmet in an attempt to punish the opponent; and (3) no player shall deliberately use his helmet to butt or ram an opponent.[7]

Since the landmark anti-spearing rule came into effect, the National Football Head and Neck Registry data clearly show a decrease in fracture, subluxation and dislocation of the cervical spine at both the high school and college level. In 1975, the season before the rule changes were initiated, the cervical spine fracture, subluxation or dislocation injury incidence was 6.5 per 100 000 and 29.3 per 100 000 players at the high school and college levels respectively.[8] In the following 8 years, a gradual decline in cervical spine injury rates occurred. By 1984, the rate of fracture, subluxation or dislocation was 3.9 and 6.7 per 100 000 for high school and college students respectively (Figure 1).[8] A similar trend occurred for permanent cervical quadriplegia between the period 1975-1984 (Figure 2).[8]

The success football has had in reducing cervical trauma and paraplegia is unfortunately not followed by Canadian hockey. University of Toronto neurosurgeon Dr. Tator reported that despite the Canadian Amateur Hockey Association's 1985 institution of prohibiting checking from behind, there has not been a decline of spinal cord injuries.[9] He attributes this to player behaviour and insufficient enforcing of the rule by coaches and referees.[9] It is interesting to note that there are about



Figures 1 and 2: *The incidence of cervical spine fractures, subluxations and dislocations and the incidence of permanent cervical quadriplegia for all levels of American football. Note a dramatic decrease in incidence following 1976, the year of rule changes. (From Reference 8, with permission).*

4 cases of quadriplegia a year in Canada due to hockey.[9] This means that on a per capita basis, hockey in Canada causes approximately three times as many quadriplegics as American football.[9]

Pathomechanics of Football Cervical Spine Injuries

As already stated, the classical concept of football cervical injuries being a result of hyperflexion injuries has largely been abandoned. Films and photographs (Figure 3) of neck injuries clearly show axial compression forces as being the primary cause of cervical cord trauma.[2,4] In the course of a collision in football,"... most of the forces are dissipated by the energy-absorbing capabilities of cervical paravertebral muscles through controlled lateral bending, flexion and extension."[10] However, when the neck is slightly flexed, the cervical spine becomes straightened and loses it normal lordotic curve (Figure 4). With this loss of cervical lordosis comes the loss of energy absorption. The cervical spine assumes the characteristics of a segmented column.[4,10] Torg states, "The bending motion is precluded in response to axially directed impacts and the energy input is transmitted to and absorbed by the spinal structures."[10] The spine buckles at some point and failure occurs in the flexion mode. This mechanism is universal for football, hockey, gymnastics and diving. Whether a football player is hit at the top of the helmet; a hockey player checked from behind; a gymnast landing on her head or a diver hitting a shallow bottom; the head is stopped and the trunk continues to move causing the cervical spine to be crushed.

It is interesting to note the added feature seen in gymnastic cervical spine injuries. A group of Swedish researchers discovered that almost all their cases of trampoline-induced cervical spine quadriplegia experienced "blackouts" prior to their falls.[4,11] It has been hypothesized that these blackouts may be due to brief concussions or transient occlusion of the posterior inferior cerebellar artery (Wallenberg's Syndrome) while the gymnast is suspended in the air.[11] This is the only example of an extraneous precipitant of sports-induced quadriplegia in my literature search.

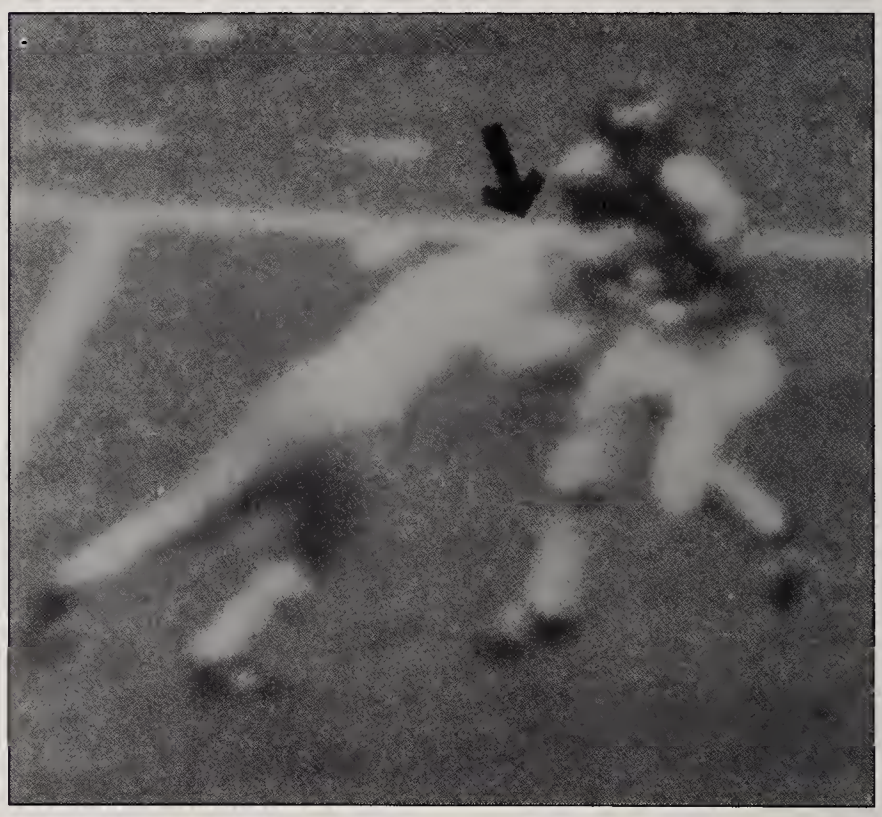


Figure 3: *Injured player (arrow) spear-tackling ball carrier with head flexed. This straightens the cervical spine and obliterates the ability to dissipate energy effectively. (From Reference 4, with permission).*

Neurapraxia of the Cervical Spinal Cord with Transient Quadriplegia

In 1986, a syndrome found in football players was receiving much attention from the athletic academic world. Torg and Pavlov reported that 1.3 per 10 000 football participants per year were being inflicted with a transient quadriplegia.[12] This syndrome appeared to be secondary to neurapraxia of the cervical spinal cord. The victim exhibited sensory changes which included burning, pain, numbness, tingling and loss of touch as well as

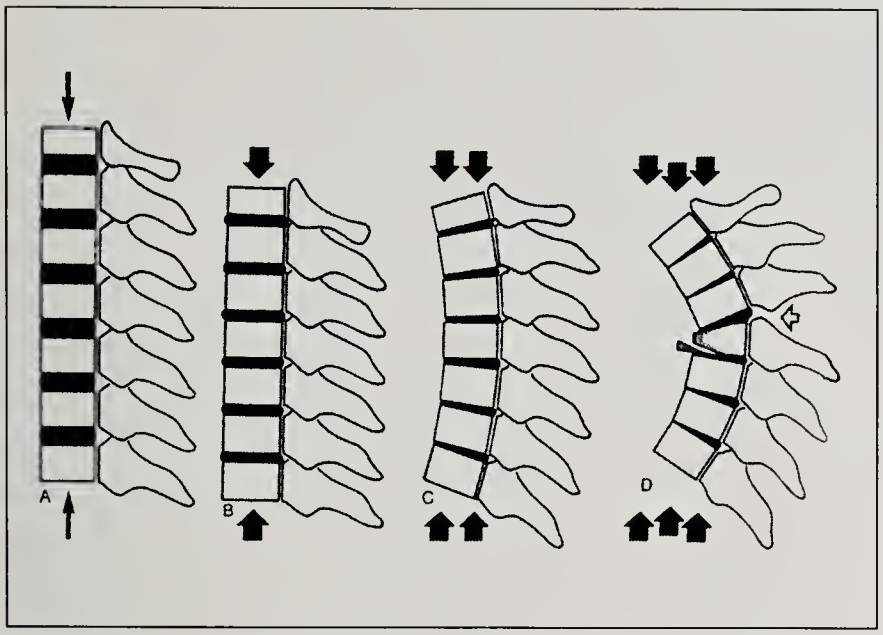


Figure 4: *Diagrammatic representation of a segmented column. (A) With 20 degrees flexion, the cervical spine straightens and responds as a segmented column. (B) With axial loads, the cervical spine compresses.(C) When the load exceeds the spine's maximum compressive potential, angulation occurs. (D) The cervical spine fails with excessive force and will buckle.(From Reference 4, with permission.)*

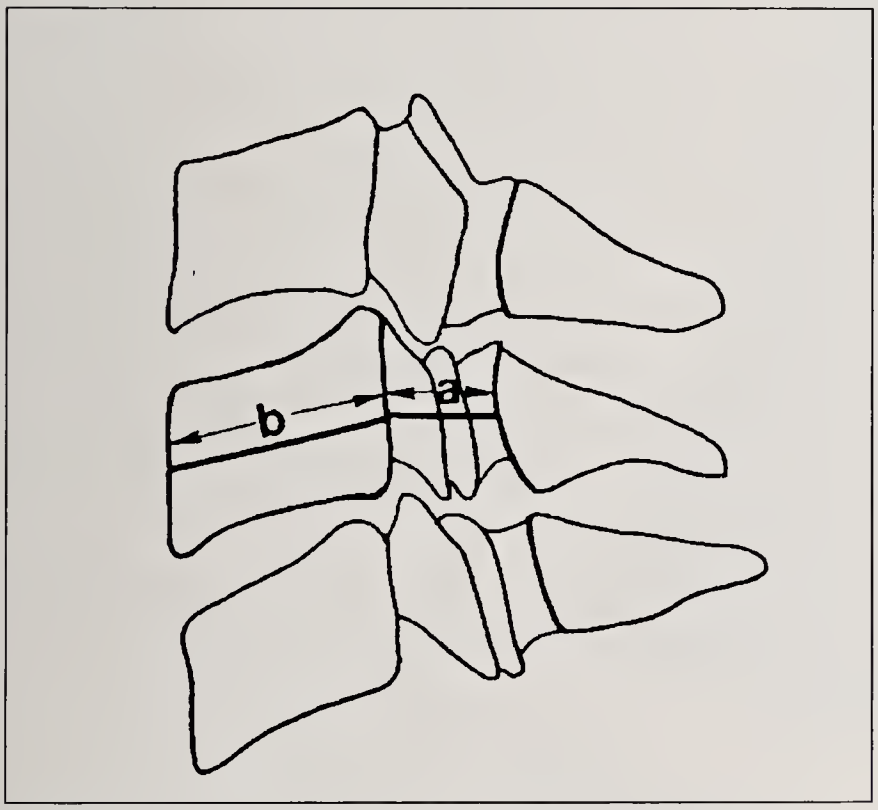


Figure 5: *The spinal canal-vertebral body ratio, or Pavlov's ratio. This ratio is the distance from the midpoint of the posterior aspect of the vertebral body to the nearest point on the spinolaminar line (a) divided by the width of the vertebral body (b). (From Reference 12, with permission.)*

motor weakness or paralysis. The episodes typically last 10 to 15 minutes but was reported in some as lasting 36 to 48 hours.[10] Pain in the cervical region is usually not present at the time of injury and the patient displays full painless motion of the cervical spine.

Cervical spine plain films were taken of 32 patients with neurapraxia. Pavlov, a Cornell University radiologist, took the distance from the posterior vertebral body to the spinolaminar line and divided this by the anteroposterior vertebral diameter (Figure 4).[12] This was done to give a relative size of the cervical spinal canal. In all 32 cases (100%), a ratio of 0.8 or less was found in at least one site of the cervical spinal cord.[12] In a group of 105 age- and sex-matched controls, only 4% had a ratio less than 0.8 at any cervical level.[10] Pavlov assigned a ratio of 0.8 or less as being suggestive of developmental spinal stenosis.[2,4,10,12]

From the above argument, one can postulate that with a diminution in cervical spinal canal size, the cord is more susceptible to be compressed with hyperextension and hyperflexion injuries. This would cause the transient motor and sensory changes. However, Torg and Pavlov strongly state that the occurrence of cervical cord neurapraxia does not predispose an individual to permanent neurological injury.[2,10,12] To achieve permanent spinal cord damage requires other key factors that will be discussed in the next section.

Spear Tackler's Spine: A Clinical Entity

The National Football Head and Neck Injury Registry showed 15 athletes in the period of 1987-1990 to have the following findings: 1)developmental narrowing of the cervical canal, i.e. a spinal canal-vertebral ratio of 0.8 or less 2)persistent straightening or reversal of the normal cervical lordotic curve on erect lateral x-rays obtained in the neutral position 3)preexisting, posttraumatic abnormalities of the cervical spine and 4)documentation of the individual having used spear tackling techniques.[10] These four factors constitute the clinical entity, spear tackler's spine. Each of the 15 individuals initially presented with complaints referable to cervical spinal cord or brachial plexus.[10] Four of the 15 patients developed permanent injuries to the spinal cord.[10] In spear tackler's spine, the straightening of the cervical lordotic curve is likely due to repetitive trauma from habitual spear tackling techniques. It has been observed that the loss of lordosis appears to be 1)a reversible reaction to muscle or ligament injury or 2)a permanent loss of the lordotic curve (Figure 6).[10,12] The straightening of the cervical segment, as discussed earlier, predisposes the player to axial load injuries. When the cervical spine is straightened, it cannot dissipate axial forces well, thus leading to injury (Figure 5). Developmental spinal stenosis and previous cervical degenerative changes are both necessary factors for the spear tackler's spine. The 15 patients in the study showed evidence of previous disc herniations, healed compression fractures and ligament laxity.[10]

Torg strongly suggests that those players who demonstrate the aforementioned characteristics of spear tackler's spine be excluded from football and other collision activities because of the risk of quadriplegia.[2,10] However, those individuals who obtain return of their normal lordotic curve i.e. muscular or tendon inflammation, may return to football.[10]

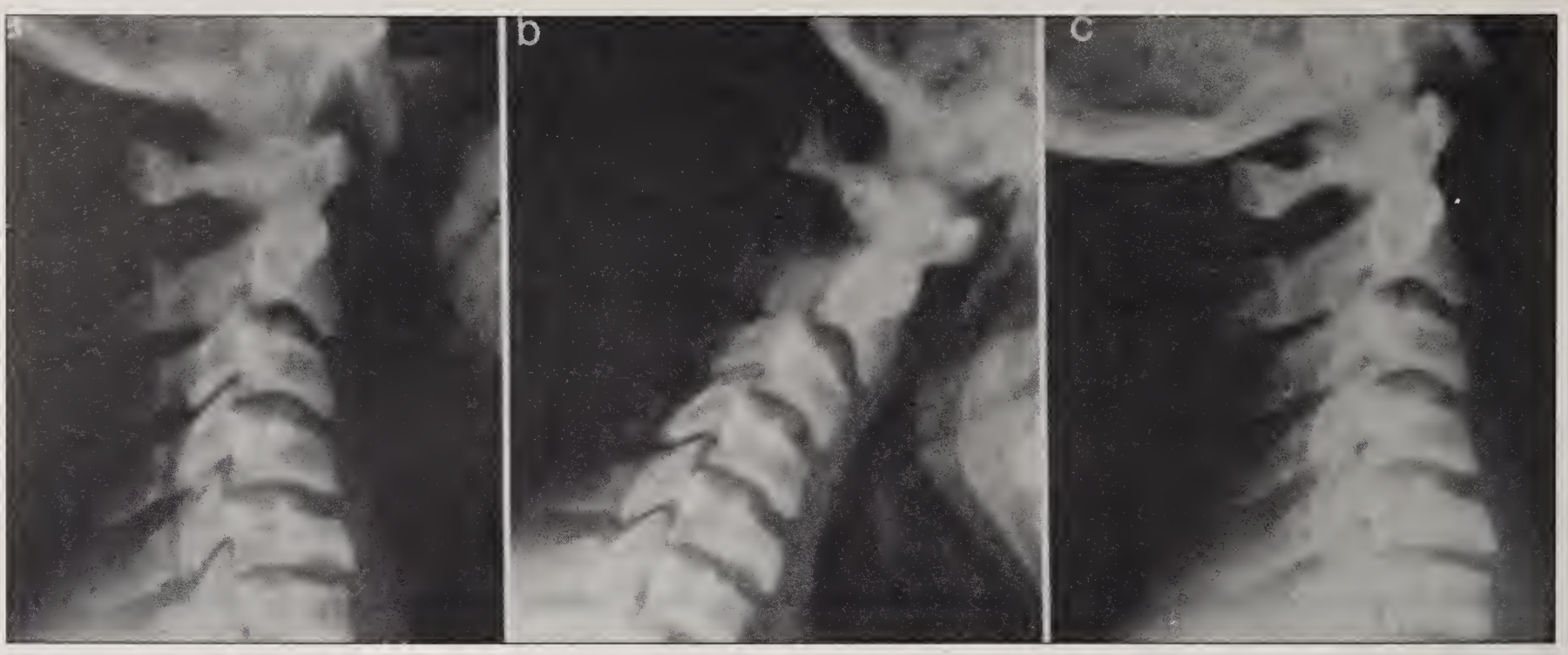


Figure 6: *Lateral cervical spine X-rays of a football player who commonly used spear tackling techniques. The (A) neutral, (B) flexion, and (C) extension views show loss of normal cervical lordosis and fixed vertebrae C2 to C6. (From Reference 10, with permission).*

Conclusion

The improved helmet-face mask developed in the 1960s allowed for a decrease in the number of intracranial injuries. However, the improved helmet served as a weapon to "spear-tackle" opponents. Until the 1976 anti-spearing rules came into effect, the number of fracture-dislocations and quadriplegics was quite significant. Spearing allowed the cervical spine to act as a straightened segmented column that could not dissipate energy effectively. It is now accepted that the axial loading force is the main contributor of cervical trauma in sports.

Clinical entities such as neurapraxia of the cervical cord with transient quadriplegia and spear tacklers's spine are newly recognized clinical entities. It has been shown that cervical cord neurapraxia is in all cases correlated with developmental cervical spinal stenosis. Spear tackler's spine presents with pain referable to the cervical cord or brachial plexus. Its four compulsory contributing factors are 1)developmental cervical spinal stenosis, 2)loss of cervical lordosis, 3)preexisting, postraumatic abnormalities of cervical spine, and 4)the use of spear tackling techniques.

"There is probably no better experimental or research laboratory for human trauma in the world than the football field..."[1] is both a bold and justifiable statement. It is an opinion widely held by many in the sports medicine world. Football has clearly enhanced our knowledge of cervical spine injuries and pathomechanics. Similarly, sports medicine has clearly changed the shape of today's football practices, allowing for substantially safer play.

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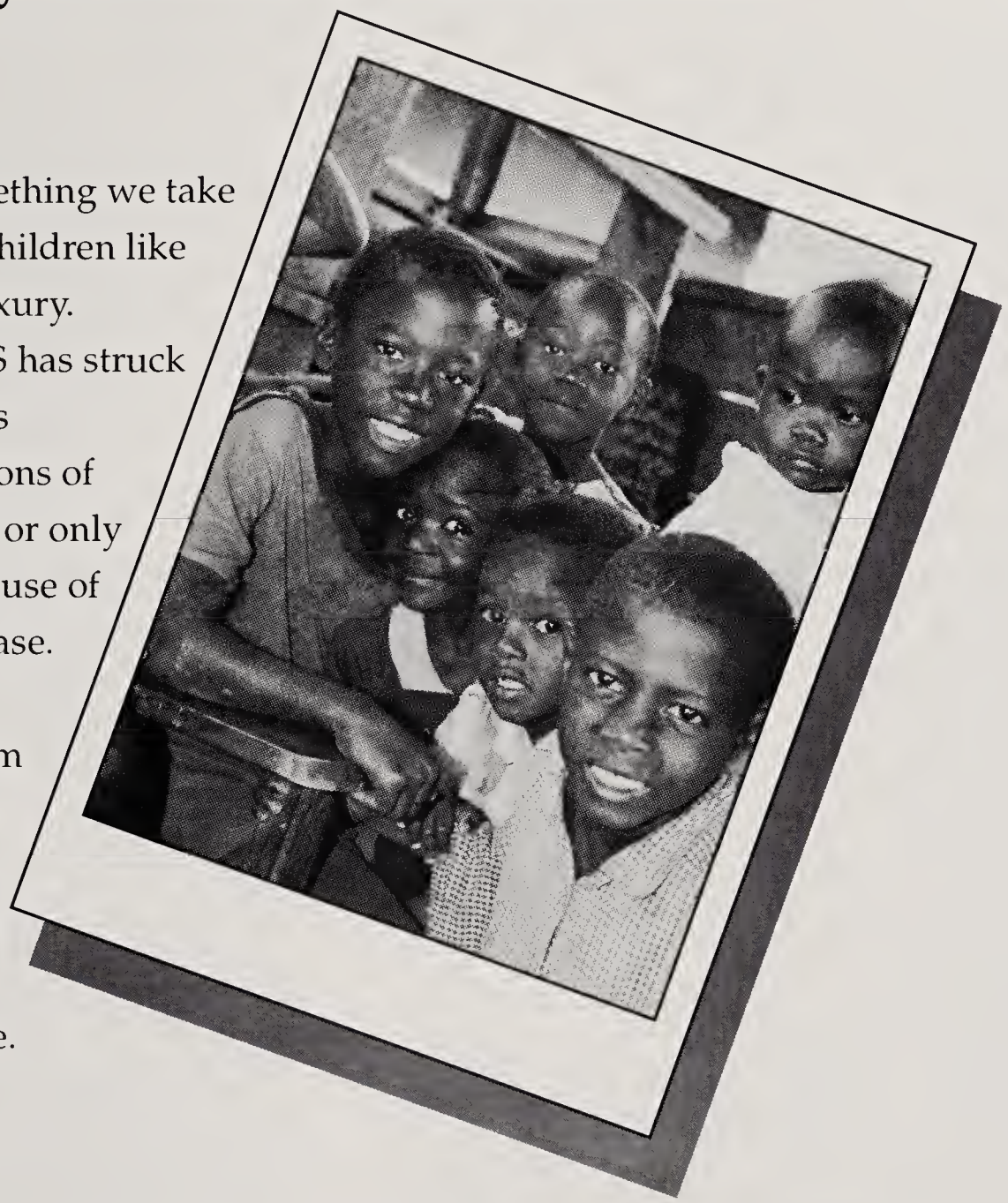

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Case 1

Lorne E. Rotstein, M.D., F.R.C.S.(C), F.A.C.S.

Clinical History

Miss L.M. is a 17 year old black female with a 3 month history of progressive left sided otalgia and increasing trismus. Dental pathology was suspected by her family physician and she was therefore referred to an oral surgeon. No obvious pathology was found in her oral cavity but a 2.5 cm node was palpated in the apex of the left posterior triangle of the neck. The oropharynx and larynx could not be adequately visualized because of her trismus.

The patient was subsequently referred to a Head and Neck Surgeon for further investigation and treatment. At that assessment, the only additional finding was of decreased sensation in the V2 distribution.

A CT scan of the head and neck was performed and is shown in Figures 1 and 2.

Figure 1: Image of a CT scan of the head showing the lesion: *axial view.*

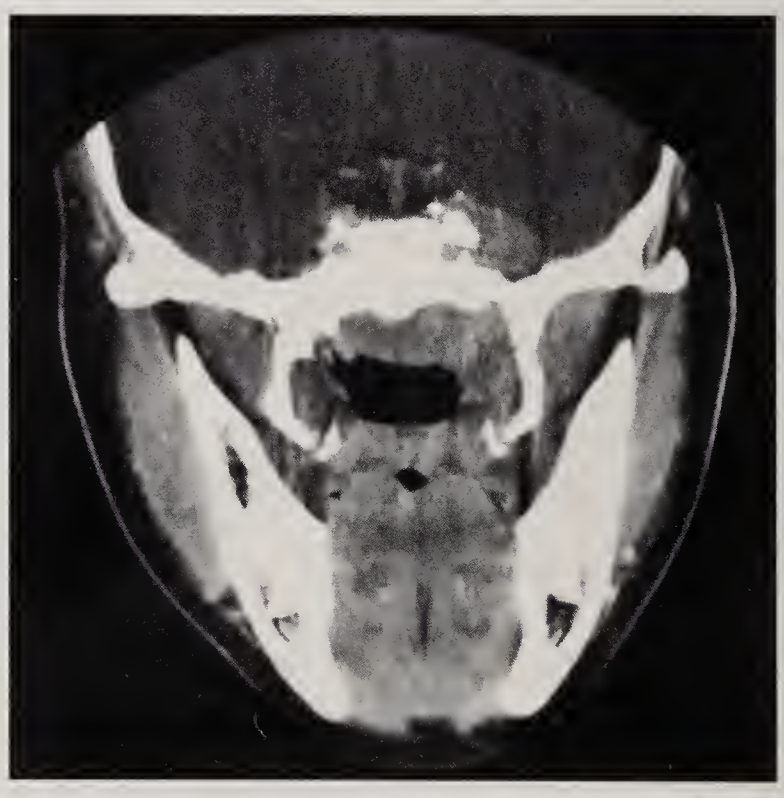


Figure 2: Image of a CT scan of the head showing the lesion: *coronal view.*

What is the differential diagnosis of this lesion?
Why does the patient have trismus and left otalgia?

Dr. Rotstein is a Staff Head & Neck Surgeon, Surgery of the Head and Neck, General Surgical Oncology, The Toronto Hospital.

Case 2

Allan E. Gross, M.D., F.R.C.S.(C)

Clinical History

A 64 year old female underwent an uneventful right hybrid total hip replacement using a one piece uncemented acetabular cup (with a metal shell and polyethylene insert) with a cemented femoral head. She did well until 18 months postoperatively when she fell landing directly on her right knee and experienced pain in her right hip. Over the next 3 months she remained painfree however experienced a clunking and "vibration" from the joint which worsened prior to reevaluation 2 years postoperatively.

On examination at that time there was an audible "clunking" from the joint with movement, although she demonstrated a good range of motion which was painless. The leg had shortened 1 cm.

Radiographs preop (Figure 1), shortly after hip replacement (Figure 2), and 2 years postop (Figure 3) are shown. Also a photograph of the PCA one piece metal-backed uncemented acetabular cup is presented.

Figure 1: Radiograph of right hip: *preop.*

Figure 2: Radiograph of right hip: *shortly following hip replacement.*

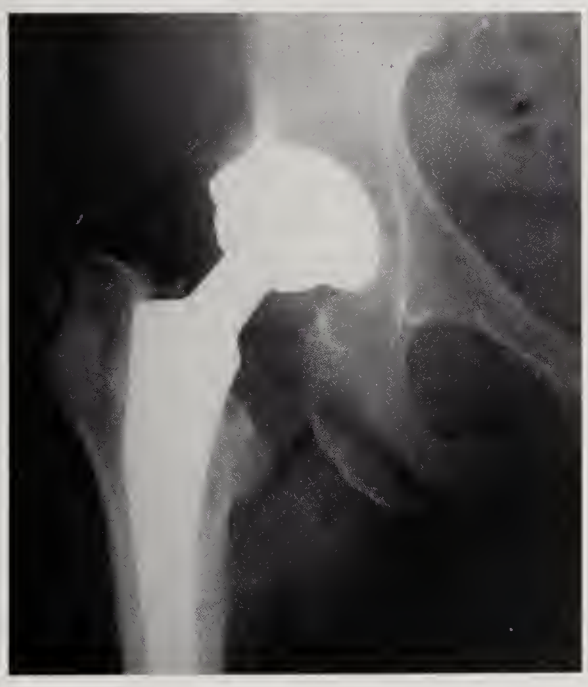


Figure 3: Radiograph of right hip: *2 years postop., note the asymmetrical position of the head within the acetabulum.*

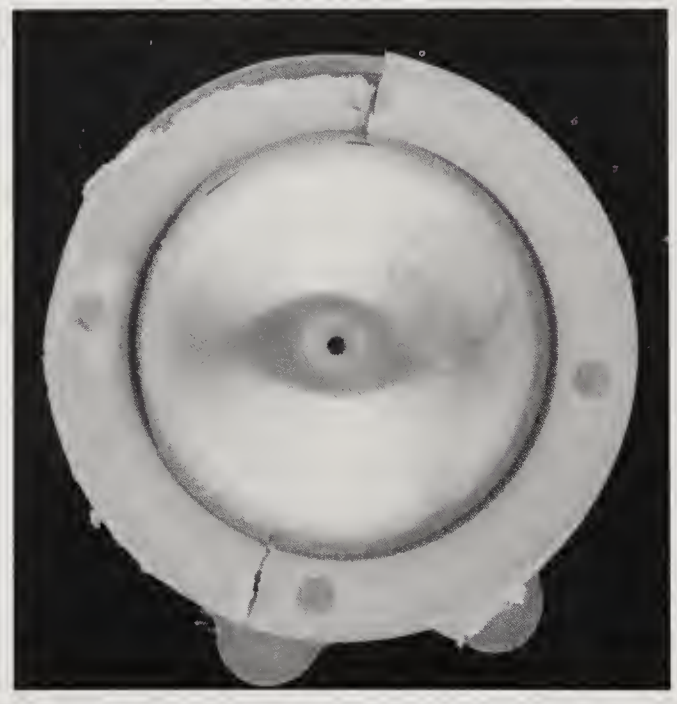


Figure 4: Photograph of uncemented acetabular cup with a metal shell and polyethylene insert along with its femoral head.

What is the most likely diagnosis?

Dr. Gross is a Professor in the Department of Surgery, University of Toronto and a Staff Orthopaedic Surgeon, Mount Sinai Hospital.

Case 3

Fred Brenneman, M.D., F.R.C.S.(C)

Clinical History

A 25 year old male was stabbed in the lower lateral chest on the left side 3 months ago. At that time he had a normal CXR and normal physical examination. He now presents with left lower chest pain on inspiration. He has no other symptoms.

He has slightly decreased air entry on auscultation of his left chest base, but no other abnormal physical findings.

A CXR, upper abdominal plain film and a CT scan of his chest are provided.

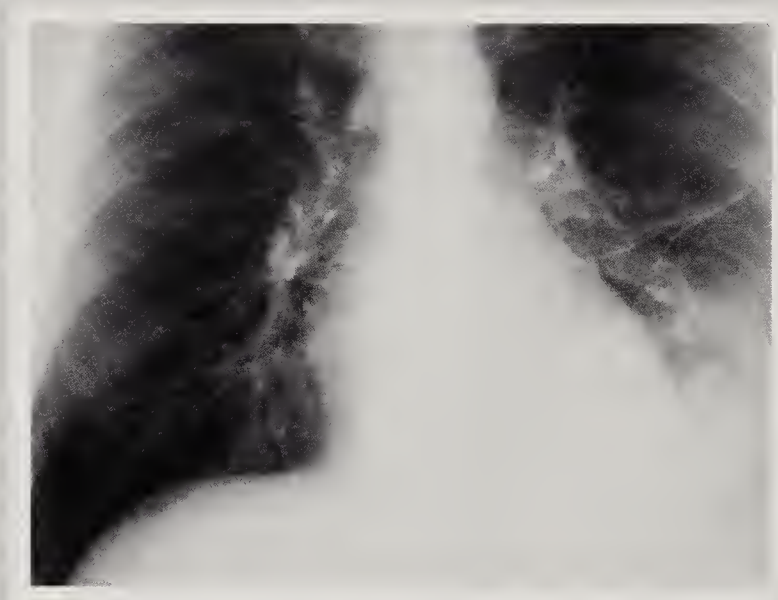


Figure 1: Chest X-rays: *PA view*

Figure 2: Upper abdominal plain film

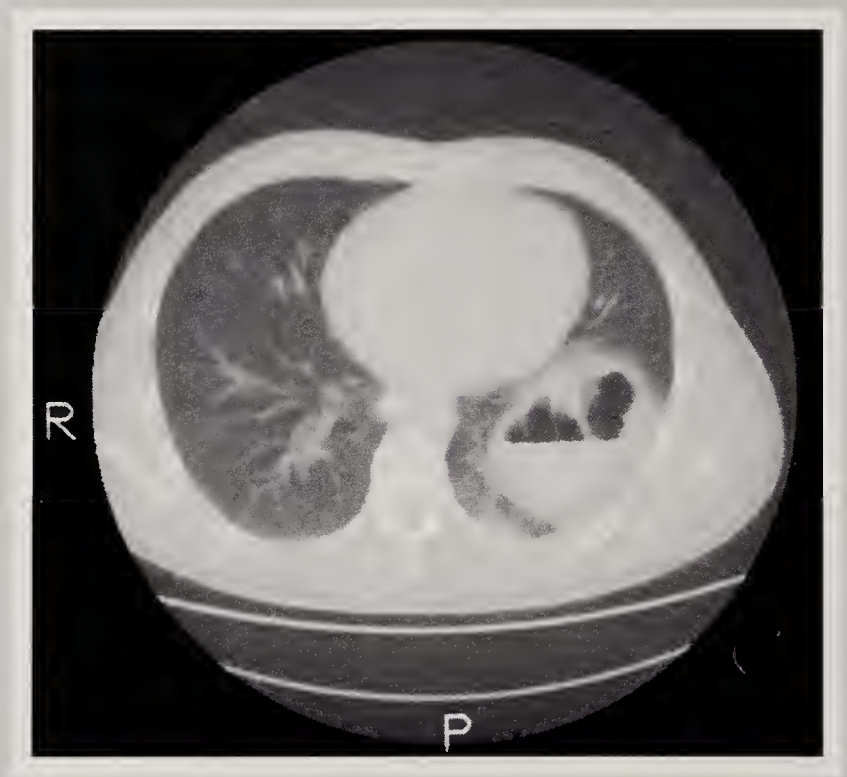


Figure 3: CT scan of chest: *axial view*

What is the most likely diagnosis?

Dr. Brenneman is a Staff Trauma and General Surgeon in the Department of Trauma and General Surgery, Sunnybrook Health Science Centre.

Case 4

Anthony Ralph-Edwards, B.Sc., M.D., F.R.C.S.(C)

Clinical History

A 72 year old male was transferred from another hospital one week after sustaining an anterior wall myocardial infarction. His course had been relatively uncomplicated until the morning of transfer when he developed recurrent chest pain associated with shock, congestive heart failure and a new systolic murmur.

A Swan-Ganz catheter was passed, high pulmonary artery pressures with v-waves were found. Right atrial oxygen saturation was 55% while the mixed venous saturation (pulmonary artery) was 80% measured simultaneously. The patient was stabilized with the placement of an intra-aortic balloon pump and inotropes. Left heart catheterization with ventriculography was performed to clarify the diagnosis (Figure 1).

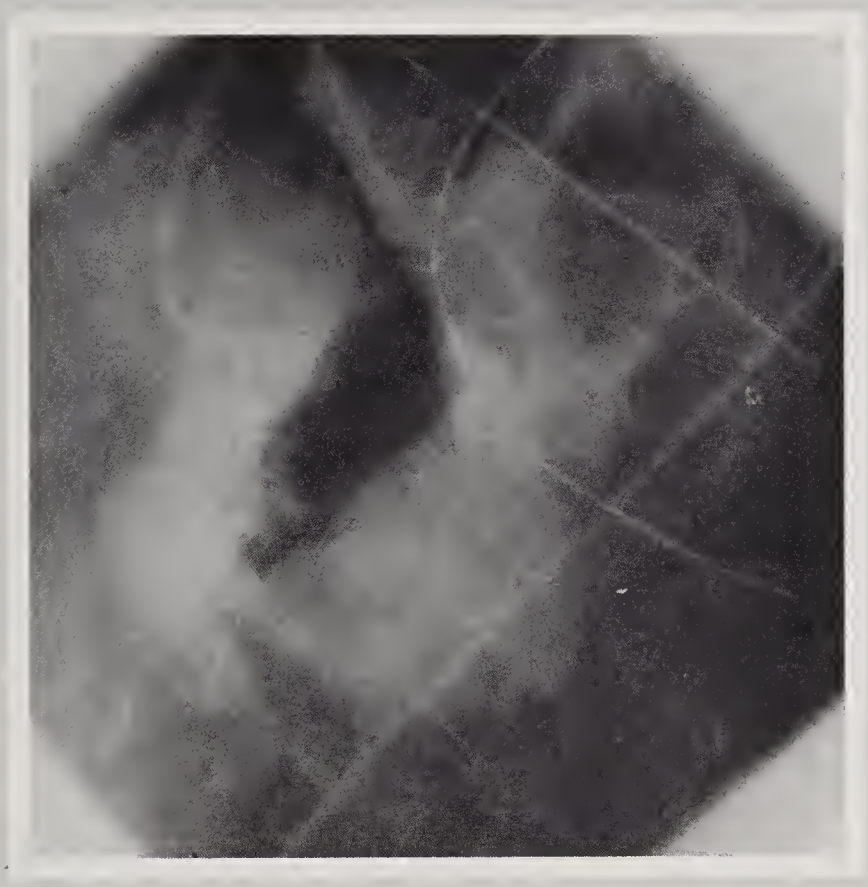


Figure 1: Left ventriculogram

What is the most likely diagnosis?

Dr. Ralph-Edwards is a Cardiovascular Surgeon, Division of Cardiovascular Surgery, The Toronto Hospital.

Case 5

Anthony Ralph-Edwards, M.D., F.R.C.S.(C)

Clinical History

This patient is a 64 year old male with a two year history of stable exertional angina. Over the weeks preceding hospitalization angina episodes became progressively more frequent, precipitated by less activity. He would also experience shortness of breath and wheezing associated with his bouts of chest pain. His past history is significant in that he has a ten year history of hypertension, documented hypercholesterolemia and is a smoker.

O/E during episodes of chest pain demonstrates elevated jugular venous pressure, bilateral basal crepitations on chest auscultation and a systolic murmur best heard at the apex of the heart.

An echocardiogram showed normal left ventricular function with no valvular abnormalities. The coronary angiogram is shown (Figure 1).

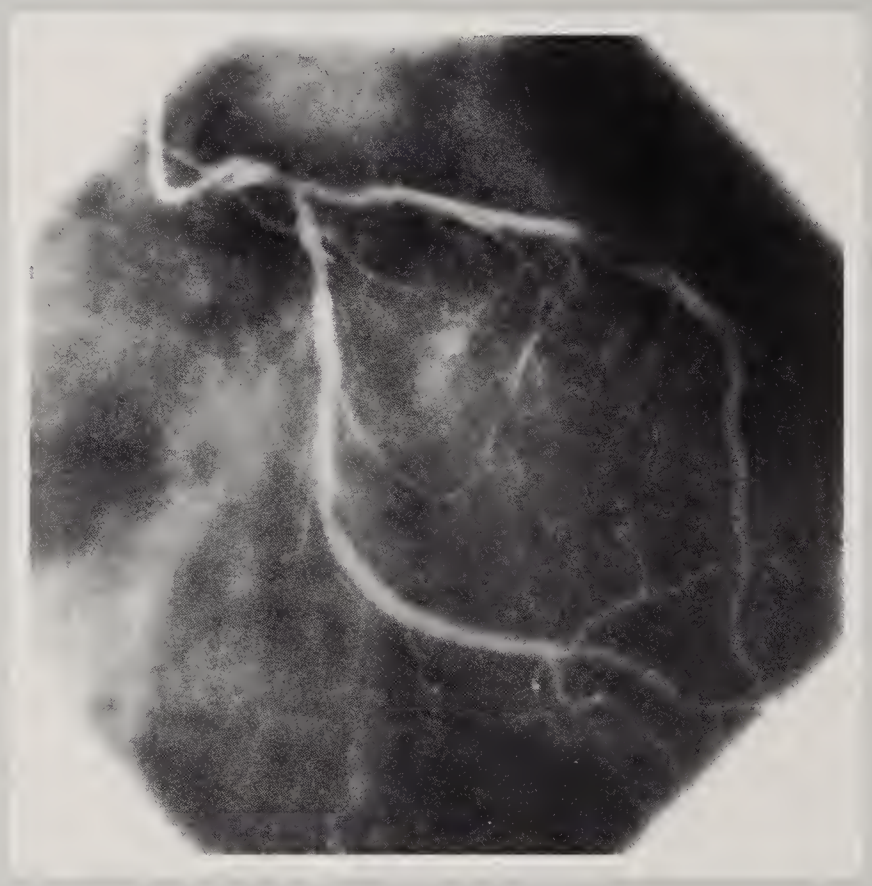


Figure 1: Selective injection of the left main coronary artery: *note severe stenosis of the distal left main coronary.*

What is the cause of episodic congestive heart failure in this man?

Dr. Ralph-Edwards is a Cardiovascular Surgeon, Division of Cardiovascular Surgery, The Toronto Hospital.

Case 6

John J. Murnaghan, M.D., F.R.C.S.(C)

Clinical History

A 35 year old male complains of significant left hip pain for the past 10 years. He developed an ache in his left groin while driving to New Jersey ten years ago. he has developed increasing pain and a limp. He experiences significant pain at night and while climbing stairs. He is no longer able to play hockey. There is no history of fever or chills. He has not taken steroids in the past. He has no bowel or eye problems. He has not had skin rashes or urethral discharge.

On examination, he is a healthy looking male, walks with limp. Negative trendelenberg sign. Leg length are equal. He flexes his hip to 120 degrees with no internal or external rotation. Abduction is limited to 20 degrees. His neurovascular status is normal.

His x-rays indicate the diagnosis.

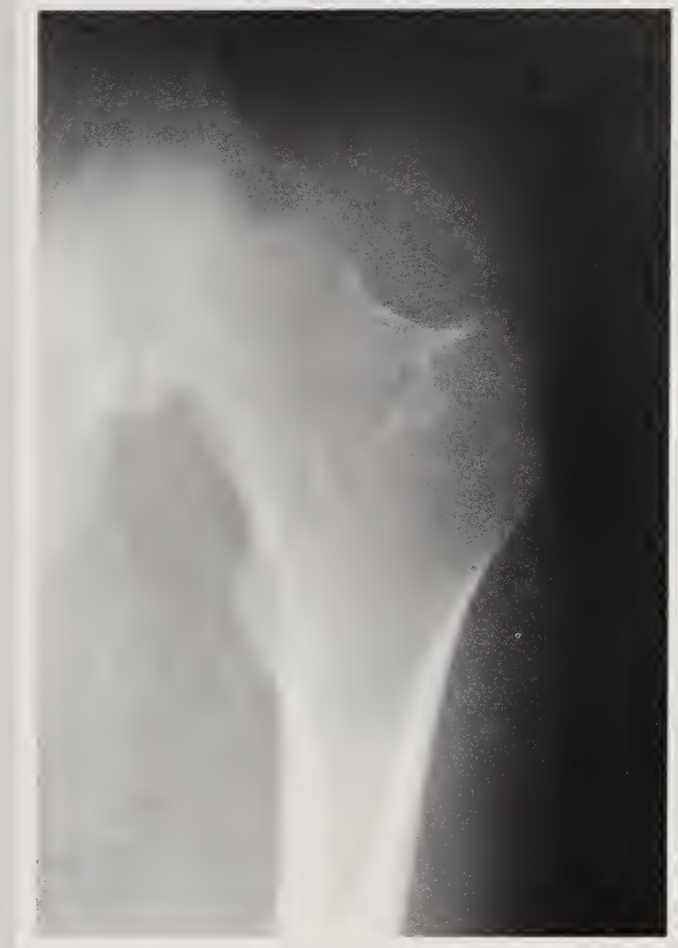


Figure 1: Radiograph of his hip left hip joint

What is the most likely diagnosis?

Dr. Murnaghan is an Assistant Professor in the Department of Surgery, University of Toronto and a Staff Orthopaedic Surgeon, The Wellesley Hospital.

ANSWERS TO QUICK DIAGNOSIS: *(UTMJ Volume 72, Number 3, May 1995)*

Infectious Diseases Rounds

Case 1

Diagnosis: **Sporotrichosis**

Case 2

Diagnosis: **Breast Abscess due to Staphylococcal infection**

Case 3

Diagnosis: **Histoplasmosis**

Predisposing factors: **AIDS/Geography**

Clinical Electrocardiography. A Simplified Approach

5th Edition
Ary L. Goldberg, and Emanuel Goldberg
Mosby-YearBook Inc.
1994
341 pp.
$54

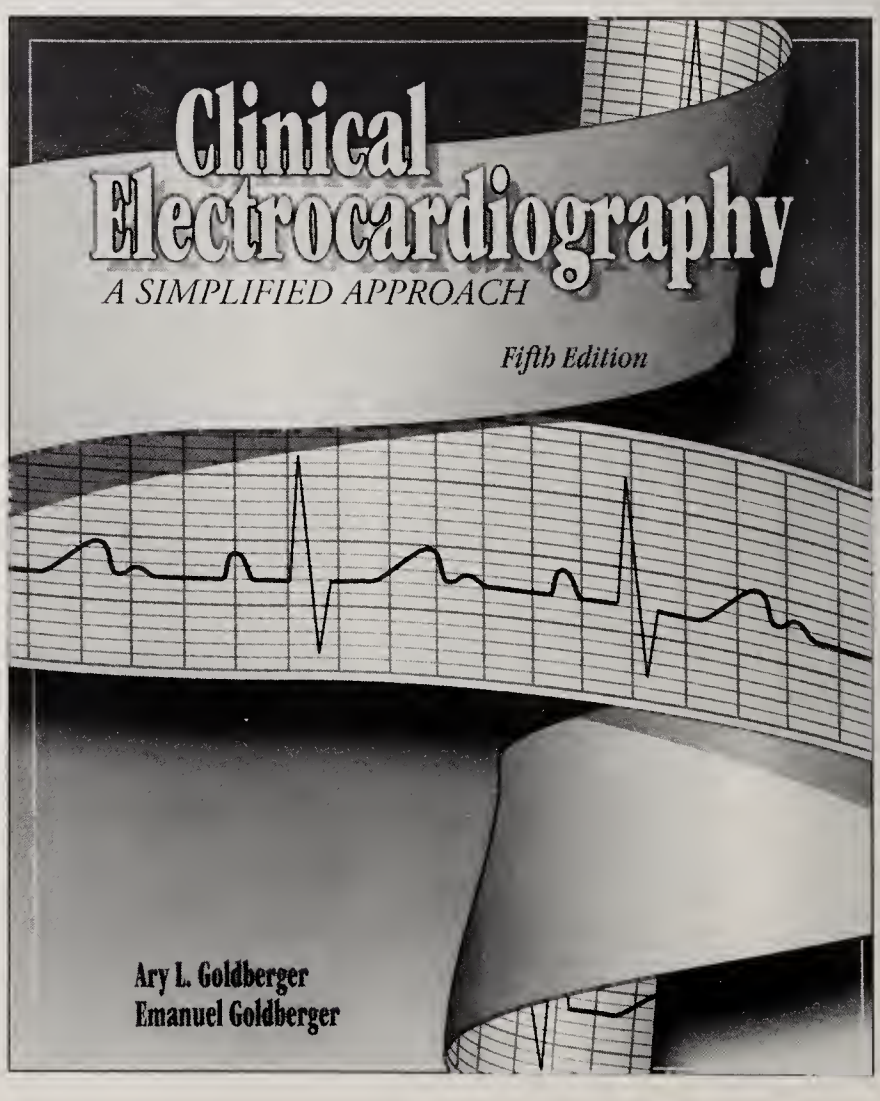
Buying a new book at an introductory level during medical school is often a frustrating experience because there are so many to choose from. I shall add to this confusion by introducing Clinical electrocardiography by Ary and Emanuel Goldberg. This book was written specifically as an introduction to electrocardiography (ECG) and assumes no previous instruction on the subject. The book is based on a series of lectures given by the authors, one of whom was the inventor of the augmented leads in 1944. It is simple in its organization and is structured to provide step-by-step instruction in ECG.

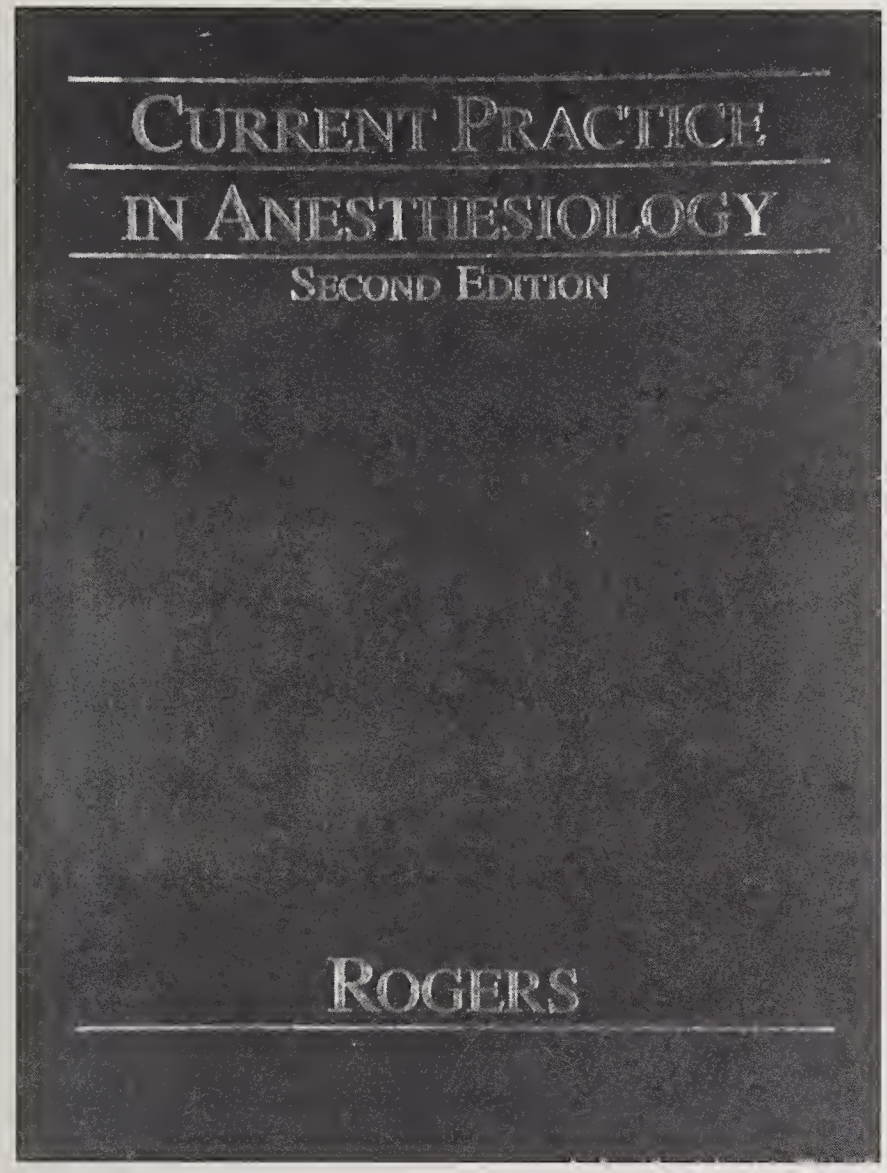
Clinical electrocardiography consists of three parts. The first two describe basic principles, patterns, and rhythm disturbances. The last part provides a wide range of self-assessment tests followed by answers with explanations. Each chapter ends with a brief summary and a few review questions which focus on the key concepts described in that chapter. This greatly facilitates learning.

The book touches on many important aspects of ECG, including general limitations and uses, introduction to pacemakers, pediatric ECG patterns, etc. Every ECG pattern description is followed by a section entitled "Clinical Significance". This makes it easier to not only understand the clinical relevance of a specific ECG finding, but to guide the reader's future research of a relevant topic. Another significant advantage of this book over other similar editions is that the authors briefly discuss general management, treatment, and the changes in ECG patterns and dynamics. The quality of the black and white diagrams and ECG samples in Clinical Electrocardiography are similar to that of photocopied material. This makes it difficult to read. This however, can be viewed as an advantage since they approximate the quality of printed material often received on the ward.

The authors stated that their aim was to "present ECG as it is used on the hospital wards, in the outpatient clinics, and in the intensive care unit, where recognition of the normal and abnormal patterns is only a starting point in patient management". In my opinion they definitely succeeded. Clinical ECG provides all the necessary basics in an easy-to-read manner, it emphasizes not only a pattern recognition, but also a differential diagnosis as well as etiology, treatment, and possible adverse effects.

Vsevolod Perelman, M.Sc, (9T7)





Current Practice in Anaesthesiology

2nd Edition
Mark. C. Rogers
B.C. Decker, An imprint of Mosby-Year Book Inc.
1990
536 pp.

Current Practice in Anaesthesiology is the combined effort of 125 contributors from all over the United States. Its goal was " to provide fresh insight into the areas of anaesthetic practice" in a complete and comprehensive manner.

This book has 19 chapters. Topics range from preoperative and postoperative considerations to descriptions of anaesthesiological nuances which may be encountered in different surgical and medical specialties. This book also has separate chapters on pain management, outpatient anaesthesia, monitoring techniques and data management. Current Practice in Anaesthesiology thus covers a wide range of applications, and reflects the opinions of the leading experts in the field.

There is significant range in the quality of the chapters in terms of readability, comprehensiveness and use of visual aids. Overall, the book reads satisfactorily, but does not give the impression of a well-organized text. In many chapters there is unnecessary repetition of basic information which could have been covered in a separate section devoted specifically to fundamental principles. On the other hand, some chapters dealing with the procedures in specific anatomical sites (ophthalmology, paediatrics, neurosurgery, etc.) dedicate a few paragraphs to review relevant anatomy, which I find very appropriate. Such reviews however, are not found in the chapters related to vascular surgery, genitourinary operations, or thoracic surgery.

There are far fewer pictures and diagrams than would have been ideal, especially in the chapters which discuss regional or local anaesthesia, nerve blocks, or intubation techniques. This may be due to the fact that the authors were catering to the more differentiated reader who may not require specific descriptions of such procedures.

Each chapter concludes with a helpful list of suggested readings in the field. Unfortufnately, most of these references are from the late 80's. The new edition will undoubtedly present an updated list.

There are interesting sections on non-medical pain management, data management and organisation of anaesthetic services. The book however, omits discussion of anaesthetic equipment and their safety features, or of common mistakes in anaesthesia and ways to prevent or correct them.

This book, in its present format, is more appropriate for those with experience in the field. It is not a textbook or manual of anaesthesiology, but may serve as a good companion to one. Better integration between the chapters as well as more visual aids for a quicker and easier reference would improve the quality of this book.

Vsevolod Perelman, M.Sc, (9T7)

Medicine, rationality, and experience: an anthropological perspective

Byron Good
Cambridge University Press
1994
242 pp.
$17.95

In 1956 Horace Miner published in American Anthropologist an account of the "body ritual of the Nacirema." He described, among many other fascinating practices, those which occur in the "*lati pso*," the temple of the medicine men. Here, for example, "vestal maidens...insert magic wands in the supplicant's mouth or force him to eat substances which are supposed to be healing. From time to time the medicine men come to their clients and jab magically treated needles into their

flesh...Excreta are used by a diviner to ascertain the course and nature of the client's sickness." Miner's prank is still amusing today, provoking us to view familiar practices from a critical perspective. Presciently, he wrote it when the study of medicine as culture was not yet a major scholarly field. Today, however, there are over 1,700 members of the Society for Medical Anthropology, as Byron Good points out in the introduction to Medicine, Rationality and Experience. In this book, Good presents several of his own field studies in the area. The collection is an excellent example of how much medical anthropology has developed in the past forty years, and how it can contribute to a better understanding of ideas about health, illness, and healing, both in our own society and in others.

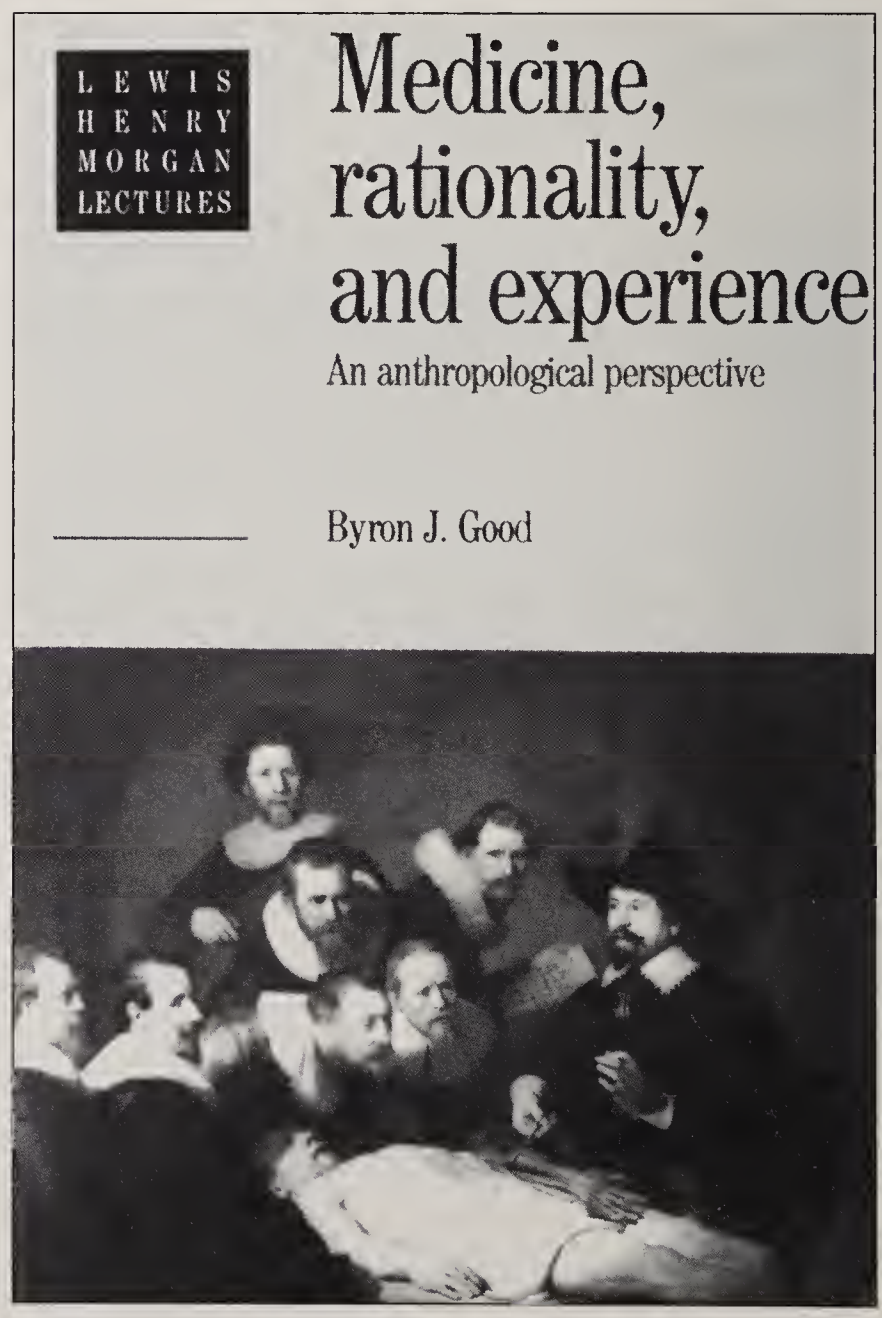
For example, in one chapter which will be of special resonance to physicians and medical students, Good, a professor at Harvard Medical School, interviewed students there about their experience as they navigate the curriculum. In presenting their reflections, he shows how they are initiated into a new culture. This is a process which "requires an extraordinary effort" on their part, as they must learn to see the interior of a person not as "thoughts, experiences, personality," but instead as an exclusively biological world. They must learn to "turn on" and "turn off" this way of seeing. For example, when first doing anatomy dissections a student might be having a conversation outside of class and think, "all of a sudden...if I took the scalpel and made a cut right here, what would that look like...And that's a frightening thing." Later, on the wards, they must learn to write and speak of patients in a way which reflects the categories - chief complaint, history of present illness, etc. - with which medicine "construct(s) a person as a patient, a document, and a project." This way of viewing people is also very removed from the students' prior experience, and "virtually every student remembers the pain of telling a story poorly and enraging a resident or attending."

Throughout the book, one of the author's main tenets is that, although we should not dispute the many explanatory and therapeutic achievements of Western biomedicine, we should not view it as the single correct representation of illness. If we make this mistaken assumption, then we tend to judge other beliefs about illness according to how well they correspond to the scientific explanation, and to stop there. Good argues that this allows only a very incomplete understanding of patients' illness experiences.

A more thorough understanding requires the realization that each patient's beliefs about illness are part of a rich web of meanings. These associations are formed through both medical and lay influences in the patient's cultural background, as well as his or her unique life experiences. For example, Good interviews a California woman who is a Jehovah's Witness and grew up on a farm - for her, her rectal bleeding is suggestive of tumours and bodily imbalance, which are in turn associated with "toxins," a "filthy environment," wickedness, and "sects" which "prostitute the truth." A series of interviews with Turkish epileptics further shows how ideas about the onset and course of an illness are entwined with surrounding events in the patient's life. It reminds us not to generalize too much when dealing with patients whom we may see as being from the same "culture." Finally, the case of "Brian," a chronic pain sufferer, represents an illness which is entirely beyond the power of Western biomedicine to explain, and shows the patient struggling on his own to give meaning to his experience.

Although, according to its foreword, the book stems from a series of public lectures given by the author, it can be at times abstruse. Particularly in the more theoretical sections where words are used in a philosophical or sociological sense without explanation. This will unfortunately limit the audience for this interesting book. On the other hand, one of the strengths of the book is its many fascinating references to other studies on diverse aspects of different illnesses throughout the world. Those doing further study in almost any area of medical anthropology, international health, public health, or social medicine will probably find something of interest in the extensive bibliography and notes at the end of the volume.

Michael Kiang, A.B., (9T7)



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Reinders FX, Chalin C, and Meier HMR. Alzheimer's disease: Understanding the patient and caregiver. UTMJ 1993; 70(2): 16-20.

For further details, see CMAJ 1985;133:443-448.

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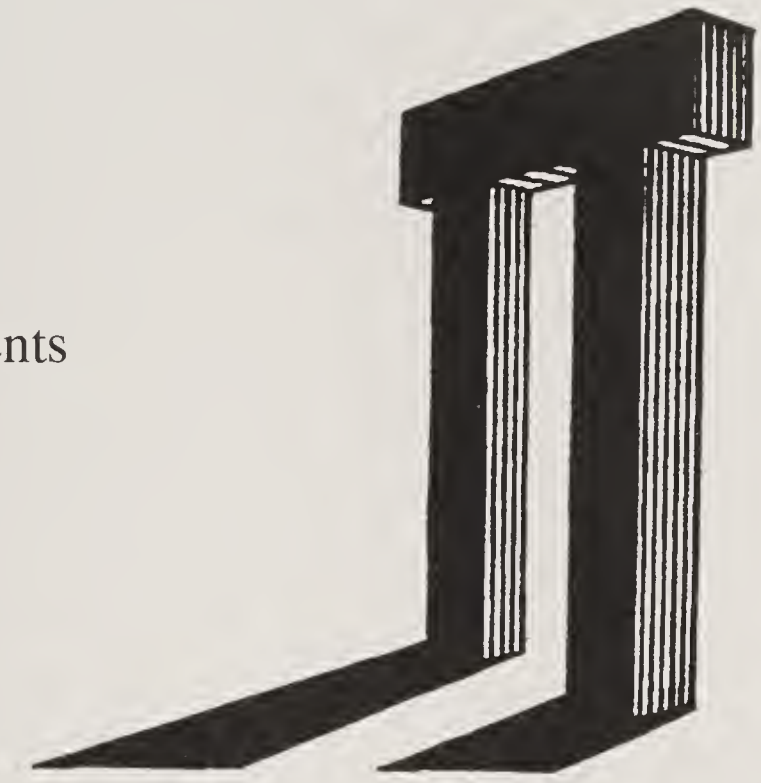

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